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ACKNOWLEDGEMENT

This booklet was written by James W. Warren in March 1971 in the form of a loose-leaf notebook for use by Fish and Wildlife Service personnel and other fish culturists in the North Central Region. Popular demand necessitated a major revision of the format in May 1972. Subsequent editorial changes and up-dating of all or portions of the material led to new editions printed in March 1978, June 1980 and September 1981.

This publication serves as the textbook for the "Introduction to Fish Health" shortcourse presented by the Hatchery Biologists of the North Central Region's Fish Disease Control Center located at the National Fishery Research Laboratory in La Crosse, Wisconsin.

TREATMENT NOTICE

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DEPARTMENT OF THE INTERIOR
U.S. FISH AND WILDLIFE SERVICE

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Washington, D.C. 20402

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DISEASES OF HATCHERY FISH

James W. Warren

U.S. Fish and Wildlife Service
Fish Hatchery Biologist's Laboratory
Genoa, Wisconsin

Much of the information in this text has been adapted from U. S. Fish and Wildlife Service fish disease leaflets, recently published books, and other articles on specific disease problems. Some of the material may go beyond the current needs of hatchery operations but it is included to meet future needs and to provide a ready reference source for hatchery workers with a developing interest in the diseases of fish.

This publication is only a guide. It cannot provide the details and experience necessary for the diagnosis or confirmation of each and every disease problem encountered. Fish disease specialists should be contacted for assistance whenever disease problems persist or losses are severe.

BACTERIAL DISEASES

Bacterial diseases occur in nature, some are caused by bacteria which are obligate fish pathogens which cannot survive indefinitely in water in the absence of susceptible host fishes. Other bacteria, commonly found in soil and water, are facultative fish pathogens which become pathogenic when fish are crowded, injured, or suffer from environmental stresses (Wedemeyer, et al, 1976). Highly virulent strains of facultative fish pathogens may behave as obligate pathogens. In nature fish are able, in most cases, to seek the best living conditions available. In the hatchery they must live under the conditions we impose. Frequently hatchery fish populations may be weakened by a wide variety of fish cultural stress conditions such as crowding, poor nutrition, handling, parasites, low oxygen, undesirable temperatures or pH's, and accumulated waste products, which lower their ability to resist infection. Meanwhile the virulence of bacteria may be increasing. When susceptible fish are exposed to virulent bacteria and the environmental conditions favor infection, a rapid increase in the death rate can be expected. When this occurs in nature, severe losses may be undetected because fish may be eaten by predators or drop unnoticed to the bottom. On the other hand, when infections kill hatchery fish the increased death rate is often one of the first changes seen.

Many bacterial diseases of fish can be treated successfully with appropriate therapeutics, others cannot. Prevention of bacterial diseases through good fish cultural practices is the best control measure.

Infected fish should not be transferred from hatchery to hatchery and water supplies should be maintained free of fish life. If a bacterial infection should break out, prompt alleviation of stress conditions, together with treatment is essential. In any case, treatment success will be short-lived if the environmental conditions contributing to the outbreak are not corrected. Research in recent years has shown that large scale immunization methods are feasible for some diseases such as vibriosis and enteric redmouth. These methods are a valuable addition to the medicine chest of all fish culturists but should not be thought of as a cure-all.

The accurate diagnosis of bacterial diseases depends upon the identification of the causative organism. The material in this section briefly describes some of these procedures. The characteristics and control of the common bacterial diseases of hatchery fish is dealt with in some detail in the latter portions of this section.

Procedures for Isolating Systemic Bacterial Fish Pathogens

1. Select typical, representative specimens which are dying and show such signs as inflamed fin joints or lesions.
2. Place 3 to 5 of the best specimens in a 1000 ppm solution of Hyamine 3500 or Roccal to disinfect the outside of the fish. Remove the fish after a minute or two and wipe clean with a fresh paper towel. Further disinfect the back of the fish ahead of the dorsal fin with 75-90 percent alcohol.
3. With a scalpel, which has been disinfected in alcohol and air dried, make a crosswise cut through the back of the fish just ahead of the dorsal fin. Cut downward through the backbone and kidney but do not cut the intestine. This technique works well on fish 4 or 5 inches in length or longer. Small fish may be opened for sampling by opening the body cavity, being careful not to cut the intestine.
4. Sterilize an inoculating loop by flaming and allow it to cool. Collect a bacterial sample from the kidney by inserting the loop into the kidney $\frac{1}{4}$ to 1 inch and rolling the loop a few times. A second sample may be collected from the opposite section of kidney exposed by the cross-cut. The posterior portion of the kidney is often the richest source of organisms, but the anterior portion is larger and is satisfactory for most purposes.
5. Inoculate TSA (Trypticase Soy Agar) slants by carefully streaking the loopful of kidney material over the length of the slant. This medium is suitable for a wide variety of organisms but is not designed to grow kidney disease, ulcer disease, columnaris, or vibrios.
6. Replace the cap and incubate at 20-22° C. for 18 to 24 hours. Slants may be packed in a padded carton and mailed immediately after inoculation.

7. For further study of the isolated bacteria, 24 hour cultures should be transferred to TSA plates. If individual colonies have grown on the slants, pick a colony with a flamed loop and streak out well in a tight back and forth pattern about $\frac{1}{4}$ inch wide at one side of the petri plate. Flame the loop and when cool streak it through this streaked area one time only and continue on with the streak, back and forth without re-entering the previously streaked area. Cover about half of the plate with fairly loose strokes. Be careful not to dig into the agar with the loop. Flame the loop again and let it cool. When cool streak through the last area streaked with a single stroke and move on to streak through the rest of the unstreaked area. It helps to rotate the plate 90 degrees between streakings. The plate should be held in the hand in a vertical (on edge) position while being inoculated. This technique will usually yield several well isolated colonies from which samples can be taken for further testing and presumptive identification.

8. It is convenient, and a good practice as well, to prepare slides of kidney material while collecting samples for culturing. With clean forceps obtain a 2 or 3 cubic millimeter pieces of kidney. Crush this material thoroughly between two glass microscope slides. Scrape most of the material off of one slide by using the other slide as a scraper. Be sure not to scrape off all of the sample; leave thick and thin areas of material on the slide. You should be able to see through most of the material.

Presumptive Identification of Bacteria

Bacteria may be presumptively identified by staining characteristics and by determining whether or not certain nutrients can be metabolized and what by-products of metabolism are produced. Quick identification can often be made through serological techniques. Slide agglutination tests show specific clumping of bacterial suspensions when immune serum is added. Fluorescent antibody techniques (FAT) have been developed for detecting and confirming bacterial kidney disease. FAT combine the simplicity of slide preparation with the specificity of serological methods to yield bacterial preparations that fluoresce a bright lime green under U-V light on the microscope. When properly prepared only the particular kind of bacteria you are searching for will fluoresce.

At the time cultures are inoculated from the fish, smears of kidney material should be prepared on glass microscope slides and allowed to air dry. Smears should also be prepared from typical colonies on 18 to 24 hour petri plate cultures.

After 18 to 24 hours, or when 1-3 mm colonies have grown, pick a sample from a typical colony, which is well isolated, to a tube containing 1 milliliter of sterile, 0.85 percent sodium chloride. Mix well. This suspension can then be used for inoculating differential bacteriological media, checking for motility and staining.

Slide Preparation

1. Use clean microscope slides with one end frosted. If dusty, clean with a soft, dry paper towel. Do not use oily slides.
2. Spread material on slide:
 - a. Bacterial culture: spread a loopful of cell suspension used for inoculating differential media on a slide; or, using a sterile loop, pick a small sample from a colony and mix with a drop of water on the slide. Spread well and air dry.
 - b. Tissue, lesion material, kidney or blood; spread thinly on a slide with a loop or another slide. Thicker material should be crushed with another slide and scraped off repeatedly until a fairly thin preparation has been made with both thick and thin areas.
3. Allow smears to air dry completely.
4. Fix smear to slide with one of the following methods:
 - a. Pass slide through a flame a few times to heat fix the material. Do not overheat.
 - b. Flood with methyl alcohol for 1 minute, drain, and allow to air dry.
 - c. Dropwise add 3 to 6 drops of methyl alcohol to slides, ignite with a match, allow to cool. Don't use any excess alcohol! There is a definite FIRE DANGER !

Staining Methods

Simple Stain -

1. Stain fixed, cooled slides with one of the following:
 - a. Methylene blue - 60 seconds
 - b. Malachite green - 30 seconds
 - c. Safranin - 90 seconds
 - d. Crystal violet - 30 seconds
2. Rinse thoroughly with water.
3. Blot gently, air dry and examine under oil immersion.

Gram Stain -

1. Stain fixed, cooled slides only.
2. Flood slide with crystal violet.
3. Add 5 drops of 5 percent sodium bicarbonate to the crystal violet on the slide.
4. After 30 seconds rinse with water but do not allow the slide to dry.
5. Add Gram's iodine to flood the slide for 10 seconds. Rinse.
6. Decolorize, with agitation, with acetone-alcohol until only a trace of blue runs off.
7. Counterstain with safranin by flooding the slide for 15 seconds.
8. Rinse, blot, air dry and examine under oil immersion.

Results: Gram positive bacteria will appear blue while Gram negative bacteria will appear pink to reddish.

Acid-Fast Stain -

1. Stain fixed, cooled smears 3 - 5 minutes with Ziehl's carbol fuchsin applying enough heat for gentle steaming. (A propane torch is quite satisfactory but make sure to entirely flood slide and keep it flooded during heating.)
2. Rinse in tap water.
3. Decolorize with acid-alcohol until only a trace of pink remains. Use 95 percent ethyl alcohol with 3 percent concentrated hydrochloric acid.
4. Rinse in tap water.
5. Counterstain with methylene blue for 30 seconds.
6. Rinse in tap water, blot dry and examine under oil immersion.

Results: Acid-fast organisms retain carbol fuchsin and are red; other organisms are blue.

Motility Test

Hanging Drop Method -

1. Place 22 mm square cover slip on flat clean surface and place a small droplet of water at each corner of cover slip and one drop in the center.

2. Sterilize a loop by flaming, cool it and pick a small portion of an isolated colony to be checked for motility.
3. Transfer bacteria on loop to drop of water in center of cover slip and mix gently without spreading the drop.
4. Invert a depression slide and center the well over the cover slip. Lower the depression slide until the corner droplets stick the cover slip to the slide.
5. Carefully and quickly turn the whole thing over so that the cover slip is on top with the bacteria in the drop of water hanging inside the well.
6. Carefully mount the slide on the microscope stage and lower the objectives almost to the cover slip. The high dry objective is usually satisfactory but oil immersion may be used. Focus on the margin of the hanging drop. Bacteria demonstrating "purposeful" movement which actually move across the field of view are motile. Simple "jiggling" is probably Brownian movement. Young cultures grown on nutritionally rich media are best for the determination of motility. Liquid media may be used for motility testing by simply transferring a drop of the grown culture to the cover slip and examining. Some liquid media types may not produce actively motile bacteria even though the organism is normally motile. A drop of the water of condensation from the bottom of a 24 hour TSA slant culture is another good source of organisms for the determination of motility.

Biochemical Tests

Dr. G. L. Bullock of the Eastern Fish Disease Laboratory at Leetown, West Virginia developed, in 1961, "A Schematic Outline for the Presumptive Identification of Bacterial Diseases of Fish" that is still of great value today (PFC, Vol. 23, No. 4, pp. 147-151). Although some methods may have changed, the procedures outlined in his flow chart can still be recommended because of their simplicity and reliability. Portions of the flow chart are described here for convenience but the reader should study the above-cited article in detail. For those interested in an expanded and updated scheme for isolating and identifying fish pathogenic bacteria see also the excellent article by Emmett Shotts and G. L. Bullock (Journal Fish. Res. Brd. of Canada, Vol 32, pp 1243-1247, 1975).

Carbohydrate Tests -

Many kinds of bacteria utilize sugars through oxidation or fermentation or both. Which sugars are metabolized and what metabolic products (acid or acid and gas) are produced in the process is important to the identification of the organism. Glucose broth is used in the flow chart format, however, other sugars and other pH indicators may be substituted in the medium if one wished to learn more about the organism in question.

1. Prepare nutrient broth as directed on the bottle.
2. Add 5 ml chlorphenol red indicator (0.4 percent chlorphenol red in 25 percent ethyl alcohol) per liter of basal medium as a pH indicator.
3. Distribute 9 ml amounts of medium to 20 mm X 150 mm screw cap tubes.
4. Insert inverted Durham fermentation tubes (10 mm X 60 mm).
5. Autoclave at 15 psi for 15 minutes with caps loose.
6. Also autoclave in the same load a 10 percent glucose (dextrose) solution. Several other sugars may also be prepared, if desired. These solutions should be bottled or tubed separately and marked for identification with a water-proof marking pen.
7. When media and sugar solution(s) have cooled, unload autoclave and aseptically transfer 1 ml of glucose solution to each tube of basal medium.
8. Inoculate liberally from a saline suspension of organisms and incubate at 20 - 22° C.
9. Observe at 24 hours for acid and/or gas production. Gas will collect in the small fermentation tube. The appearance of an acid (yellowish) reaction only at the top of the medium indicates the organism is oxidative. Acid throughout or acid throughout and gas indicates fermentation or both oxidation and fermentation.

Hydrogen Sulfide Test -

1. Prepare motility sulfide medium as directed on bottle.
2. Distribute 10 ml amounts into screw cap tubes.
3. Autoclave 15 minutes at 15 psi.
4. Allow medium to gel in a deep butt.
5. Inoculate by stabbing medium from top to bottom in a single stab. Withdraw needle through the same path.
6. Incubate 24 hours at 20 - 22° C.
7. Blackening of the medium after incubation indicates the production of hydrogen sulfide. Some reactions may take longer than 24 hours and at times only a little blackening may be found. Record these differences.

Starch Hydrolysis Test -

1. Prepare nutrient agar as directed on the bottle.
2. Add 0.2 percent soluble starch to boiling medium during preparation and mix well.
3. Distribute 10 ml amounts into screw cap tubes.
4. Autoclave for 15 minutes at 15 psi.
5. Allow medium to gel in the slanted position leaving little or no butt.
6. Inoculate by drawing a loopful of saline-suspended bacteria up the center of the slant in a single straight line. Do not zig-zag.
7. Incubate at 20 - 22° C. for 24 - 48 hours.
8. When good growth has occurred add 1 ml of Gram's iodine and allow iodine to cover entire slant.
9. Scrape bacterial growth off of a section of the growth area with a loop to allow penetration of the iodine.
10. The iodine will react with the starch in the medium and will form a deep blue or dark brown color if the starch has not been utilized by the bacteria. The appearance of a colorless zone (in young cultures especially) in the growth area indicates the starch has been utilized and constitutes a positive test.

Cytochrome Oxidase Test -

The test for the presence of cytochrome oxidase is most easily performed through the use of commercially prepared cytochrome oxidase test papers. Pathotec-CO[®] test papers are recommended and should be stored in the refrigerator.

1. With sterile forceps remove a fresh test strip from the vial.
2. With a sterile loop pick a loopful of bacterial growth from an 18 - 24 hour pure culture. Growth from any nutritious agar slant or plate is satisfactory. Growth from the starch agar slant is a good source if the slant has not been treated with iodine used in the starch test.
3. Apply the bacteria to the slightly colored area of the test paper.
4. The fairly prompt appearance of a deep blue color in the test paper in the area where the growth was applied is a positive test for cytochrome oxidase presence.

2, 3-Butanediol Test -

The 2, 3-Butanediol test, if positive, will confirm the presence of Aeromonas hydrophila as virtually no other organisms commonly encountered in the aquatic environment will produce a positive reaction.

1. Prepare the following test medium:

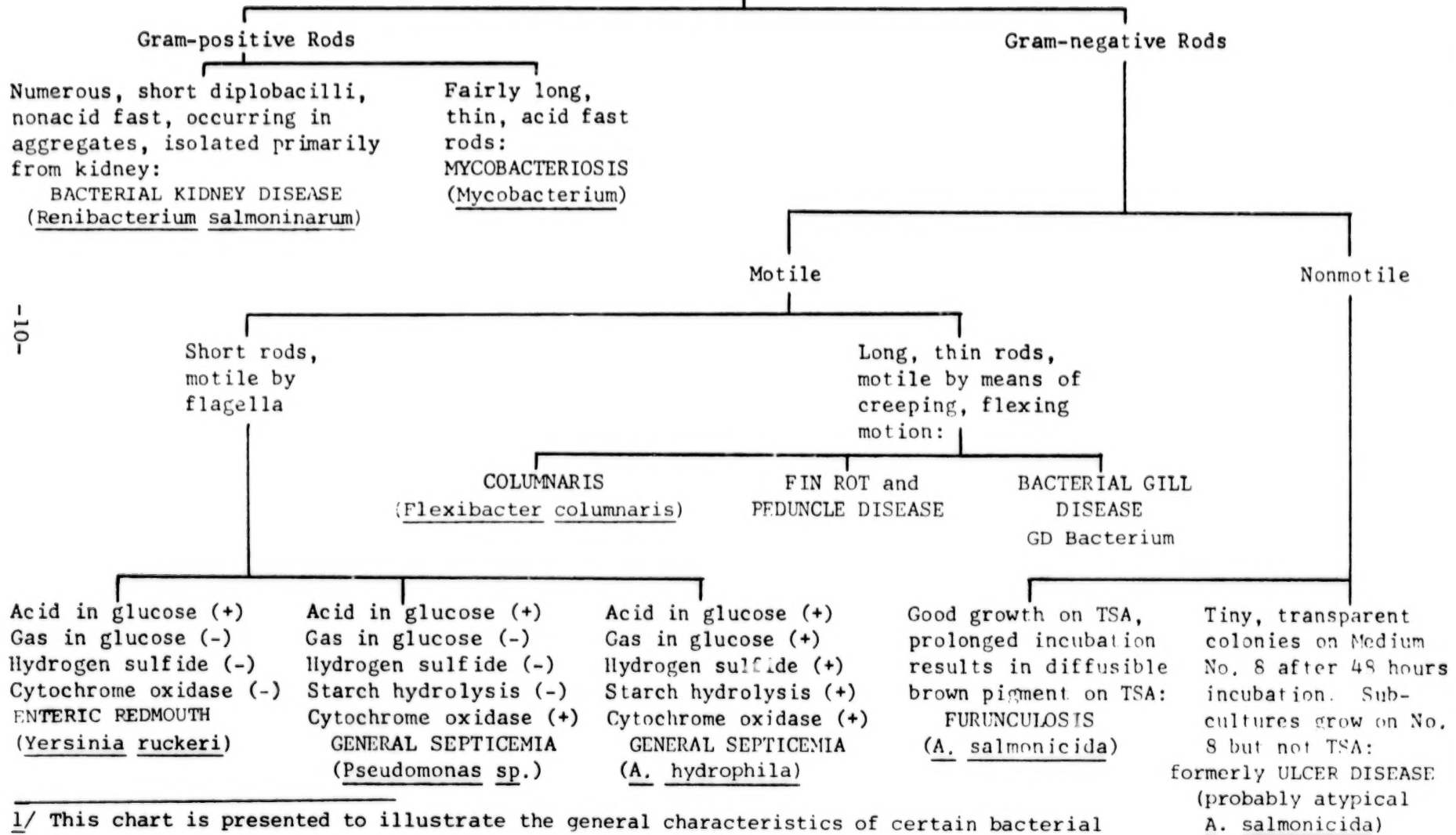
Nutrient broth	8.0 grams
Dibasic potassium phosphate	5.0 grams
Distilled water	1.0 liter

2. Distribute medium in 4.5 ml amounts into screw cap tubes.
3. Autoclave for 15 minutes at 15 psi with caps loose.
4. In the same load autoclave a 15 percent glucose solution. Allow the load to cool before removing from autoclave.
5. Aseptically add 0.5 ml of glucose solution to each tube of basal medium.
6. Inoculate liberally from a saline suspension of bacteria.
7. Incubate at 20 - 22° C. for 24 - 48 hours or until fairly turbid.
8. Test for the presence of 2, 3-Butanediol as follows:
 - a. Pipet into each grown culture 1.0 ml of 2.3 percent periodic acid (solution should be stored in brown, glass stoppered bottle).
 - b. Shake tube and let stand $\frac{1}{2}$ hour at room temperature.
 - c. Add 1.5 ml of piperazine solution (25 grams of piperazine hexahydrate dissolved in 99 ml distilled water then add 1.3 ml 87 percent formic acid). Then add 0.5 ml of 4.0 percent sodium nitroprusside solution (prepared daily).
9. Development of a distinct blue within 2 minutes is considered positive for the production of 2, 3-Butanediol while a pale green is considered negative.

Drug Sensitivity Tests

Small discs, impregnated with various concentrations of a wide variety of therapeutic drugs are commercially available and easy to use. By placing these discs on specially prepared media plates the drug will diffuse into the media. If the drug inhibits the growth of the bacteria being tested, a clear zone free of bacterial growth will appear adjacent to the discs. If, on the other hand, the drug has little or no effect upon the growth of the organism the bacteria will grow right up to the disc. These results must be interpreted with caution as positive inhibition of the bacteria on sensitivity plates does not guarantee success of the drug in treating fish as the drug might not be absorbed from the gut or other complications might arise.

BACTERIOLOGICAL PRESUMPTIVE TESTS ^{1/}
(Primary Isolation - Pure Culture)



The methods for preparing sensitivity tests are as follows:

1. Prepare a turbid saline suspension from a young culture of the bacterial isolate to be tested.
2. Soak a sterile cotton swab in the saline suspension and lightly press out the surplus on the inside of the tube.
3. Carefully paint the bacteria over the entire surface of a Muller-Hinton media plate in a tight back and forth pattern. Rotate the plate 90 degrees and paint it again.
4. Discard the swab into a container of disinfectant.
5. With sterile forceps, remove drug discs from their vials and place on the surface of the agar. Discs should be spaced about 3/4 to 1 inch apart and about that far from the edge of the plate. Don't move discs once they touch the plate and press each in place rather firmly.
6. Incubate the plates upside down at 20-22° C. for 18-24 hours.
7. Examine the plates for zones of inhibition and record the results. Good large (10-20 mm diameter) zones with clear centers indicate possible value of that drug as a therapeutic. No zone or zones with isolated colonies growing within them indicate probable drug resistance. Look for the clearness of the zones in particular. Some drugs diffuse through media better than others. Different drugs are designed to inhibit different kinds of bacteria so don't expect all drugs to give positive results. Indications of drug resistance to approved therapeutics such as sulfamerazine and terramycin should be reported to your fish disease specialist.

Furunculosis

Introduction

Furunculosis was first reported in the United States in 1902. Since then, most trout and salmon hatcheries have either been infected or exposed at one time or another. At times epizootics have been threatened the very existence of fish culture. Today furunculosis is enzootic in many hatcheries. Severe outbreaks occasionally occur but due to advances in fish culture, sanitation and drug therapy, most are controllable. Outbreaks of drug resistant furunculosis are an increasingly important problem.

The disease is caused by the bacterium Aeromonas salmonicida and is characterized by a generalized bacteremia with focal lesions in the muscles. Many cases have been observed in which these lesions are absent, however. Furunculosis bacteria enter the fish through a scratch or through the digestive tract. The bacteria then become localized and challenge the defensive mechanisms of the fish. The bacteria produce a toxin which is released when some of the bacteria are lysed by the host. This endotoxin destroys the inflammatory response in the fish leaving it unprotected. A generalized infection then develops that kills the fish. The furunculosis bacterium is capable of infecting many species of fish, including suckers and non-game fish which inhabit hatchery water supplies, and thereby serve as reservoirs of infection.

Signs

External -

1. Swollen skin "blisters" filled with pinkish or reddish fluid, which have a purple, reddish or iridescent blue color are common on salmonids with a chronic infection.
2. Hemorrhaged fin sockets, especially the left pectoral, and frayed dorsal fins are common.
3. In acute cases lesions do not develop but the gills may be attacked. The only apparent sign in cases of this sort is white or pale pink gills. Fish are actually bleeding to death through the gills. To demonstrate this, press the gill covers and watch for blood coming from the gills of lethargic fish. Death may be rapid in this sort of infection and only a few sick fish may be seen at any one time in spite of the high death rate.

Internal -

1. Examine the area around the heart; when few signs are present, the pericardium which walls the heart off from the main body cavity is usually filled with bloody fluid and is inflamed.
2. Inflamed, red spots may be found in the lining of the body cavity and especially on the visceral fat.
3. The spleen, normally dark red in color, will often be a bright cherry red and swollen.
4. In chronic cases, occurring during periods of very low water temperatures, pus-filled lesions may be found in the kidney.
5. The lower intestine is often highly inflamed and a bloody discharge can be manually pressed from the vent.

Diagnosis

Confirmation of furunculosis as the cause of an epizootic depends upon the isolation of A. salmonicida from typically affected fish. Furunculosis should be suspected, however, when some or all of the above signs are observed and water temperatures average 56° F. or higher. For convenience a few of the key characteristics of the furunculosis bacterium are listed here but, the flow chart, on page 10, should be consulted to see where A. salmonicida fits with respect to other fish-pathogenic bacteria.

Some Characteristics of A. salmonicida

1. Gram negative, non-motile rod
2. Cytochrome oxidase positive
3. Acid and gas from glucose
4. 2,3-butainediol negative
5. Produce reddish-brown pigment in TSA culture medium.
[Some atypical (achromogenic) strains may not produce this pigment]

Epizootiology

Range -

Furunculosis has been frequently reported from all parts of the world except Australia and New Zealand. With increased production of salmonids in these countries, the disease will soon be diagnosed if it has not been diagnosed there already. Furunculosis is enzootic in many hatcheries and is so widespread that no natural waters with resident fish populations should be considered free of this disease.

Season of Occurrence -

The incidence pattern of furunculosis generally follows the seasonal temperature pattern. By far, the most cases of furunculosis occur in July. The number of July outbreaks is nearly double that of any other month. The number of cases reported drops sharply in August, possibly indicating increased resistance in the remaining fish population or removal of most of the susceptible fish by mortality.

Incubation Period -

Acute cases of furunculosis have short incubation periods of from 2 to 4 days and few signs are apparent. Chronic cases, the sort that are usually encountered below about 55° F., may have an incubation period of one week to several weeks depending upon the water temperature. Latent cases may develop during low water temperature periods. When water temperatures rise, these flare up with severity and the appearance of typical disease signs.

Susceptibility and Resistance -

Exposed fish apparently respond to furunculosis and form protective antibodies. Some fish become immune carriers of the infection. Suckers and non-game fish in the water supply should be considered as likely reservoirs of infection. Under conditions favoring the organism and disfavoring the fish such as high water temperatures for the species and low oxygen levels, furunculosis may break out in virtually any freshwater fish population including warm-water species. The disease cannot tolerate sea water and does not spread from fish to fish in sea water unless a fish eats an infected fish.

In the late 1970's a non-pigment forming (achromogenic) strain of A. salmonicida was found to be the cause of serious outbreaks of disfiguring skin lesions in commercially reared goldfish. Furunculosis has also caused significant mortalities in smallmouth bass and in tiger muskies in New York state.

Immunization of fish populations has been partially researched and mass immunization methods are being developed. There is hope of immunizing fish against furunculosis but no widely applicable, reliable system has been made available for use in the field.

Methods of Control

Prevention -

1. Avoid hatchery to hatchery transfer of fish.
2. Maintain springs and water supply systems free of fish, including scrap-fish. Hatcheries utilizing stream water should, if possible, block scrap-fish from migrating above the water intake during their spawning run.
3. Avoid handling fish during, or for 2 to 3 weeks previous to, the expected time of usual outbreak.
4. Fish eggs should be disinfected even though furunculosis has not been shown to occur in fry or fingerlings hatched from contaminated eggs.

Therapy -

1. Feed sulfamerazine at 5 grams per 100 pounds of fish for 10 to 15 days or until losses have reached normal for 5 days. A 10 gram level, designed for administration through the wet diets, has long

been recommended but the more efficient dry diets assure better consumption. Drug resistance against the sulfa drugs is quite common.

2. Terramycin, fed in the feed at 3.0 grams per 100 pounds of fish is generally accepted as the standard treatment for furunculosis in salmonids. Treatment should run 10 to 15 days or for 5 days after losses reach normal.

Aeromonas and Pseudomonas Infections (General Septicemia)

Introduction

The bacteria belonging to the genera Aeromonas and Pseudomonas may, under certain adverse environmental conditions, cause serious septicemic infections. These bacteria are common soil and water bacteria found in most natural waters. For example, Aeromonas hydrophila, which is often isolated from fish, is found in large numbers in water bearing a heavy organic burden. These bacteria break down organic matter of all kinds and do much to "freshen" surface water.

Stresses caused by high water temperatures, low oxygen levels, accumulation of waste products and crowded fish populations are essential predisposing factors which lead to disease outbreaks attributed to the bacteria Aeromonas hydrophila and Pseudomonas fluorescens. These organisms are opportunists capable of developing increased virulence under optimum conditions. Work by Dr. Fred Meyer at Stuttgart, Arkansas has shown that A. hydrophila infections peak during June, July and August. He attributed these increased losses to the stresses caused by frequent low oxygen levels which weaken the fish.

Signs

External -

1. Superficial, shallow-grayish, red circular or irregular skin ulcers.
2. Distended abdomen which is filled with a slightly opaque or bloody fluid (dropsy).
3. Possible popeye caused by fluid behind eye.
4. Cyprinids may have deep furuncles which may break out to the surface producing deep craters.
5. Fins and fin joints may be inflamed.

Internal -

1. Opaque or bloody fluid in abdomen around organs.
2. Kidney may be swollen and soft.
3. Liver may be pale or greenish.
4. Lining of body cavity may have small hemorrhagic spots.
5. Intestine and vent are usually inflamed, swollen and may contain yellowish mucus or a bloody discharge. Note: Acute infections may cause high mortalities with few signs.

Diagnosis

The determination that mortalities are being caused by A. hydrophila or P. fluorescens must begin with a recognition of probable adverse environmental conditions of some sort. Next would be the observation of moribund fish and the detection of some of the signs listed above. Bacteriological isolation of A. hydrophila or P. fluorescens and the identification of these bacteria through presumptive tests would then be meaningful and could be interpreted as the likely cause of the mortality. The presence of these bacteria does not necessarily mean mortalities will occur. However, if handling, temperature, low oxygen or other stresses predispose fish to infection then the detection of these organisms, consistently, in moribund fish is sufficient evidence to consider these organisms to be the cause of the difficulty.

The flow chart on page 10 shows the relationship between these bacteria and other important bacterial fish pathogens. Listed below are selected characteristics that help distinguish A. hydrophila from P. fluorescens:

<u>Characteristic</u>	<u>A. hydrophila</u>	<u>P. fluorescens</u>
Motility	positive	positive
Glucose	acid & gas	acid
Starch	positive	negative
2,3-Butanediol	positive	negative
Cytochrome Oxidase	positive	positive
Gram stain	negative	negative

Epizootiology

Range -

The greatest losses to general septicemia or infectious dropsy have occurred in carp culture in Europe. The organisms are common world-wide in waters rich in organic material. Diseased fish or frogs, or fish and frogs which have recovered but still carry the infection, are reservoirs of infection. In the United States these diseases are most common in warm-water ponds but flare up from time to time in trout and salmon operations.

Season of Occurrence -

Disease outbreaks are most numerous in June, July and August in warm-water fish culture and virtually non-existent in winter. Cloudy weather may lead to low oxygen levels when pond blooms are heavy which in turn leads to stress and outbreaks of septicemia.

Incubation Period -

Depending upon extrinsic factors, outbreaks may occur almost simultaneously with adverse fish cultural conditions or up to several weeks later. Assuming the organisms to be present continually, the time required for an epizootic to develop would be largely temperature dependent and could be 5 to 7 days or longer.

Susceptibility and Resistance -

Warm water fish species tend to lose acquired immunity to general septicemia over the winter months. To date the development of immunity in fish to protect against general septicemia has met with only limited success. European carp culturists have had some success with the genetic development of disease resistant strains of fish.

Methods of Control

Prevention -

1. Avoid hatchery to hatchery transfers of fish. Fish gradually develop resistance to the local strains of bacteria but may carry virulent organisms to another hatchery when transferred.
2. Provide optimal environmental conditions for the species being reared, paying special attention to the maintenance of oxygen levels and the gentle handling of fish.

- ③ 3. When sorting, handling and moving broodstock in the spring, prophylactic treatments have been helpful. Intraperitoneal injections of 10 to 30 mg per pound of body weight of aqueous chloramphenicol has reduced post-handling losses by 80-90 percent in Europe. If
- ③ 4. P. fluorescens was isolated, fish culturists have injected streptomycin intraperitoneally at the rate of 75 mg per pound of body weight.

4. Antibiotics or disinfectants should be added to the water as a prophylactic measure when fish are transported in tank trucks or plastic bags. Fish culturists have tested the procedure they happen to choose to determine its toxicity before proceeding on a large scale.
- ③ Conditions can vary widely and caution should be taken. Acriflavin, at 2-4 ppm has been used in routine fish transportation operations and may have value. Distribution tanks are a useful unit to use for the external treatment of fish for parasites and bacterial infections.

5. Selective breeding of resistant stocks of fish has promise for the long range control of general septicemia.

Therapy -

1. With the widespread use of dry pelleted feeds in the rearing of many species of fish, there developed an excellent means of treating fish. Terramycin has been effective when incorporated into pellets at the mill or coated on the feed at the hatchery. Terramycin should be fed at 3.0 grams of active drug per 100 pounds of fish per day. Sulfamerazine has also been used. It may be less effective than Terramycin but if the latter is not available and sulfamerazine is on hand it should be fed at the rate of 10 grams of drug per 100 pounds of fish per day. Treatments with either drug should continue for at least 5 days after mortalities have approached normal. Antibacterial therapy will provide only brief relief if stress conditions such as adverse environmental conditions or crowding are not promptly corrected.

Vibriosis

Introduction

The cause of vibriosis, Vibrio anguillarum is primarily a marine bacteria. It can live in brackish waters and home aquaria. Historically, vibriosis has been associated with outbreaks of red disease of eels the world over. In recent years vibriosis has become recognized as a cause of severe losses of salmon smolts shortly after their migration or transfer to salt water.

With the advent of huge impoundments for rearing salmon fingerlings to marketable size in salt water it quickly became apparent that early losses as high as 90 percent were common (Fryer, et al, 1972). Apparently the fish reared in freshwater were not prepared immunologically to cope with vibrios. Most of the vibrios isolated from moribund fish have been found to be quite similar to V. anguillarum, falling into one of three major serological groups.

Signs

External -

1. Salmonids have a "buckshot" appearance along their sides where many small hemorrhagic spots can be seen.
2. Large, bright-colored bloody lesions may be found in the skin and musculature beneath.
3. Hemorrhagic areas at the base of the fins are common.
4. Pressure on the gill covers may readily cause bleeding from the gills.

Internal -

1. Hemorrhagic spots may be found throughout the body cavity.
2. Inflammation of the intestine is common.
3. A bloody discharge may be expressed from the vent.
4. Hemorrhages may be found in the eye, with or without popeye.

Diagnosis

Identification of vibrios depends upon their isolation and separation from other bacteria. V. anguillarum is a small, Gram-negative rod which is motile by means of a single polar flagella. In many respects they could be confused with the Aeromonads or the Pseudomonads. Most vibrios will not grow well, or at all, on ordinary aqueous media but must have 1.5 to 2.0 percent sodium chloride in the medium for growth. Motility

of V. anguillarum is rapidly lost in tap or distilled water but lasts for some time in wet mounts prepared with saline. Final identification of vibrios is usually performed by testing them against a vibriostatic agent, 0/129 (2,4-diamino-6,7-diisopropyl pteridine).

Epizootiology

Range -

Vibrio infections have been reported world-wide with serious outbreaks in cultured salmonids, eels, and other brackish water species.

Season of Occurrence -

Vibrio infections occur primarily in the summer months when water temperatures warm above 50° F. and become severe above 60° F. Below 48° F. the virulence of vibrios is markedly reduced.

Incubation Period -

In natural infections the incubation period has been observed to be as short as 3 to 5 days in salmonids. Undoubtedly temperature is an important factor.

Susceptibility and Resistance -

Chum and pink salmon are especially susceptible to vibrio infections when reared in salt water ponds. Other species of salmonids such as rainbow and steelhead trout have suffered serious losses in freshwater hatchery operations. Northern pike have been lost to vibrio disease in Europe. Eels are particularly susceptible.

Methods of Control

Prevention -

1. Fish should not be stressed by handling or crowding during late spring or summer when water temperatures are high.
2. Oxygen levels should be maintained above 5 to 6 ppm.
3. Protective vaccines have been developed by John Fryer and his co-workers at Oregon State University. Bacterins which have been prepared from killed whole cells of V. anguillarum can be fed, injected, or sprayed on fish. The regimen used most often in salmon culture involves feeding an oral vaccine for the 15 day period ending about two weeks prior to the transfer of the smolts to salt water. The vaccine prepares the fish for the initial encounter with vibrios and natural contact sustains immunity.

Therapy -

1. Terramycin at 3.0 grams per 100 pounds of fish for 10 days or until normal mortalities have been reached for 5 days is the treatment of choice. Treatments will have only a limited long-term value if stress conditions cannot be alleviated simultaneously with the treatment.



2. Sulfamethazine at 5 grams per 100 pounds of fish for 10 days may be an effective alternate.



3. A combination of 3 grams of sulfamethazine and 2 grams of furoxone per 100 pounds of fish for 10 days may be of value in cases not responding well to the other drugs.

Enteric Redmouth (ERM)

Introduction

Enteric redmouth (ERM) probably kills more rainbow trout each year than any other bacterial disease. It is responsible for severe systemic infections in cultured rainbow in the Inter-mountain West and, in recent years, from Alaska to the Carolinas and from Maine to California. The spread of this disease can be traced directly to the transfer of ERM carriers from one hatchery to another. Few cases can be linked to other modes of transmission.

Enteric redmouth is caused by a Gram-negative, peritrichous rod which is highly motile in fresh cultures. The bacterium was recently named Yersinia ruckeri in honor of Dr. Robert R. Rucker, formerly Director of USFWS Western Fish Disease Laboratory in Seattle, WA, who stimulated much of the early work on this disease.

Signs

External -

Black, lethargic rainbow trout which are blinded by popeye and drift listlessly at the water surface should always be thoroughly examined for enteric redmouth. Important additional signs are:

1. The mouth and tongue are often grossly inflamed.
2. The whitish underbelly may have speckled or blotchy hemorrhages.
3. Moribund fish may have distended abdomens.
4. Hemorrhages in the fin sockets are common.
5. Heavily infected fish may bleed from the gills if the gill covers are firmly pressed.

Internal -

1. A flaccid, water-filled stomach is a common and important internal sign. Redmouth should be suspected whenever this condition is found.
2. Visceral fat may be flecked with hemorrhagic spots or may appear uniformly pinkish rather than the normally white color.
3. The lower intestine often is grossly inflamed and may contain a yellowish or bloody fluid.
4. The kidney commonly is swollen.

Diagnosis

The confirmation of the presence of redmouth disease depends upon the identification of the redmouth bacterium. Consult the flow chart to determine the relationship between the redmouth bacterium and other common fish pathogenic bacteria. For convenience, some of the major characteristics of the organism are listed here:

1. Gram-negative rod
2. Motile
3. Cytochrome oxidase negative
4. Acid but no gas from glucose
5. 2, 3-Butanediol negative.

Epizootiology

Range -

Enteric redmouth disease is enzootic in the Inter-mountain West but additional enzootic foci have been established in other areas of the United States where rainbow trout are grown. A prime factor has been the long distance transportation of infected fish. The disease is a continual problem in southern Idaho where it is believed to have originated.

Season of Occurrence -

At hatcheries where redmouth is a frequent problem, the disease is at its worst during the hot summer months even though the water temperature remains constant.

Incubation Period -

Experimental work has shown the incubation period to be 5 days at 59° F. Although probably temperature dependent, incubation periods at other temperatures are not known.

Susceptibility and Resistance

Enteric redmouth disease is a disease of rainbow trout but other salmonids should not be considered safe from infection. It is found in rainbows because few other species are reared in the enzootic areas. Rainbows are highly susceptible and losses of 55 - 85 percent are not uncommon if treatment is withheld. A commercially prepared vaccine has been developed for the mass immunization of fingerlings to help reduce the impact of enteric redmouth at hatcheries where the disease is a recurring problem.

Methods of Control

Prevention -

1. Avoid the transfer of infected or exposed fish from hatchery to hatchery.
2. Immunize rainbow trout fingerlings reared in enzootic areas when fish are 1000/pound.
3. Maintain all water supplies free of fish.
4. Maintain light population densities whenever possible, especially during the hot summer months.
5. Do not handle fish during mid-summer.
6. Maintain oxygen levels above 6 ppm at the outlet.

Therapy -

1. Terramycin, at the rate of 3.0 grams per 100 pounds of fish for 10 days or for 5 days after mortalities have reached pre-outbreak levels must be considered the standard treatment of choice. As with any therapeutic procedure, good results depend upon the prompt development of an effective drug level in the fish and the alleviation of stresses.

 2. Combination therapy has been reportedly successful. A combination of sulfamerazine and terramycin fed at the rate of 3.0 grams of each drug (6 grams total) per 100 pounds of fish for 10 days or for 5 days after mortalities have reached pre-outbreak levels. During treatment, stress conditions leading to outbreaks were alleviated whenever good results were obtained.

Bacterial Gill Disease

Introduction

Bacterial gill disease (GD) is one of the most common diseases of hatchery salmonids and is sometimes found in warm-water pond-fish. It is a superficial infection of the gill epithelium by large numbers of long, thin, filamentous bacteria of undecided identity which often leads to the clubbing and fusing of gill filaments. Distress is caused by the resulting inability of the fish to obtain oxygen and rid itself of carbon dioxide and ammonia. Gills damaged by GD bacteria are also prone to invasion by fungus. In acute cases, massive numbers of bacteria develop in a short time and the loss rate may jump from 0 to over 20 percent in one day with little change taking place in the gill epithelium. Death appears to be due to the sheer numbers of bacteria blocking normal respiration.

Metabolic products evidently are responsible for enabling GD bacteria to become established on the gills. Crowding of fish, especially small fingerlings (approximately 100/pound and smaller) seems to be a prime factor in the occurrence of bacterial gill disease. Lightly infected fish have been found to recover spontaneously if they can be moved from a pond environment to fresh running water.

Signs

1. Gross external signs may be confused with those caused by the protozoan parasite Ichtyobodo (Costia).
2. Fish first appear lethargic and some refuse food.
3. Some fish may appear to be pale in color.
4. Fish "ride high" in the water and orient with the current and space themselves equidistant from one another.
5. Increased or labored gill activity is often noticeable with the gill covers opened wider than normal.
6. Gills may appear whitened on the tips and excess mucus may be present.

Diagnosis

Confirmation of the presence of bacterial gill disease is based upon observation of the above-described disease signs and the observation of GD bacteria on the gills. Acute cases may come on so quickly that the above signs appear all within 24 hours and mortality rates may be explosive. Examination of gills will show huge numbers of GD bacteria on the gill lamellae with varying degrees of tissue damage. A slide prepared from crushed gill filaments and stained with methylene blue will greatly aid in the detection of GD bacteria during microscopic examination. The bacteria

appear as tangles of long slender rods. Numerous tangled masses of GD bacteria will be found on each slide prepared if the disease is reaching serious proportions. Microscopic examination is essential to accurately diagnose the presence of bacterial gill disease.

Epizootiology

Range -

GD bacteria are an apparently ubiquitous water organism probably of world-wide distribution. Bacterial gill disease probably occurs throughout the range of salmonids but seems especially severe in salmon hatcheries in the Pacific Northwest. Hatcheries in that part of the country often rely upon stream water for their water supply thereby possibly introducing organisms from wild fish.

Season of Occurrence -

The bacterial gill disease season coincides with the fingerling salmonid season. A 50 percent increase in cases occurs in March and the cases peak in May. By June, distribution has begun on larger fish and the fingerlings are spread into empty raceways ending the problem. A minor increase of cases occurs in August as rearing units once again become crowded.

Incubation Period -

The incubation period for bacterial gill disease varies with the strain of bacterium and the susceptibility and density of the fish population. Acute cases in sockeye salmon or chinook salmon populations may explode within a 24 hour period while chronic cases may take several weeks to develop.

Susceptibility and Resistance -

The sockeye is the salmon species most susceptible to bacterial gill disease, followed by chinook and coho. Rainbow trout and steelhead trout are apparently the most susceptible of the trout species although serious cases are common among browns. Bacterial gill disease is primarily a disease of young fingerlings smaller than about 100 fish per pound.

Methods of Control

Prevention -

1. Fingerling salmonids should be reared on fresh water until they reach at least 100 fish per pound in size. Reuse of water should be avoided if at all possible.

2. Maintain population levels as low as possible.
3. Maintain the cleanliness of rearing facilities and provide sufficient water to flush away waste products.
4. Pick mortalities and remove "pinheads" which can be reservoirs of infection.
5. Chemical treatments as described below for therapy can be applied on a routine basis during the fingerling rearing period to prevent bacterial gill disease at hatcheries where the disease frequently occurs.

Therapy -

-  1. For trout (except lake trout): The use of Hyamine 3500 or Roccal at 2 ppm of active ingredient, for 1 hour on three successive days has been effective.
-  2. For salmon and lake trout: Hyamine 1622 at 2 ppm of active ingredient for 1 hour, on 3 - 4 successive days, or Diquat at 8.4 ppm (as it comes from the bottle which gives 2 ppm active ingredients) for 1 hour on 3 - 4 consecutive days has worked well for these species.
-  3. For pond fish: Diquat has been used as a pond treatment at 0.5 to 1.0 ppm active ingredients. This treatment coincidentally controls algae which aids in improving environmental conditions. Care should be taken in the discharge of Diquat after treatment as it is a potent herbicide. At present the use of Diquat is restricted to weed control and has not been cleared for use on fish by the FDA.

Columnaris

Introduction

Columnaris is caused by the bacterium Flexibacter columnaris. This disease derives its name from the peculiar trait the bacterium has of aggregating into clumps, "haystacks", and eventually into distinctive columns. Flexibacters are long, thin soil and water bacteria that can be highly pathogenic to fish. They move about by bending and twisting flexible cells. They secrete slime as they go.

The columnaris bacterium likely enters fish through the skin and the gills. If the skin has been scratched the bacteria enter rapidly. Even uninjured gills can be attacked by virulent columnaris bacteria.

Water temperature has a major influence on the occurrence of outbreaks of columnaris. Outbreaks seldom occur at water temperatures below 55° F. but may be explosive above 65° F. Crowding, handling and other stresses should be considered as predisposing factors although some highly virulent strains of flexibacters have been found that will attack apparently healthy fish with only warm water to contend with. Serious outbreaks have occurred in the Pacific Northwest in salmon populations in the Columbia River.

Signs

External -

1. Lesions on the body begin as small circular erosions of the skin which rapidly increase in size. Many lesions are dished out, exposing the muscle. The tissue often is covered with a bright yellow slime, particularly at the margin of the lesion.
2. Lesions as described above are common on the head and in the mouth of fish. The yellowish color is produced by the bacteria and their slime.
3. Gill lesions often begin on the end of the filaments and progress toward the base. Extensive lesions will completely denude the gill cartilage. Highly virulent strains of flexibacters attack the gills causing the epithelium to separate from the capillaries beneath. The fish die of rapid suffocation. Mouth and body lesions are usually caused by the less virulent strains.

Internal -

1. Columnaris is essentially an external infection but in well advanced cases the causative organism can be isolated from the kidney and other internal organs indicating a systemic infection. Gross internal changes are not apparent in most cases.

Diagnosis

The diagnosis of columnaris depends upon the identification of typical lesions lined with massive numbers of flexibacters. Necrosis of the areas affected is of diagnostic importance, especially on the gills. This characteristic alone distinguishes columnaris of the gills from gross bacterial gill disease. Yellow slime and massive numbers of flexibacters in the lesions are also keys to columnaris detection. Microscopic examination in wet mounts is especially informative for the experienced diagnostician as the formation of columns can be observed. Simple stained (methylene blue) slides of lesion material should show huge numbers of bacteria.

Epizootiology

Range -

Columnaris probably occurs world-wide; attacking a wide variety of freshwater fish. The disease is particularly prevalent in the Pacific Northwest where it is associated with anadromous fish species. The warming of the water of the upper Columbia River, by the construction of dams, in the semi-arid inter-mountain area and the use of river water to cool atomic manufacturing and power generating facilities has been blamed for the apparent increase in losses in upstream-migrating adult salmon. Pondfish in all parts of the United States may be attacked by columnaris. Temperature of the water is the prime factor with few mortalities occurring if the water temperature is below 55° F.

Season of Occurrence -

The incidence of columnaris follows a seasonal pattern that almost exactly duplicates the seasonal water temperature curve. The number of reported cases more than doubles between June and July and this high rate continues through August. Columnaris, therefore, is a summer-time disease.

Incubation Period -

Two types of flexibacters are recognized: a highly virulent form and a form with low virulence. The various serological strains detected likely fall into one of these two groups. The highly virulent form generally attacks the gills and the incubation period may be 24 hours or less. The low virulence form usually attacks the skin, causing external lesions, but killing the fish at a much slower rate. The incubation period for this form may be from 48 hours to several weeks depending upon the water temperature. Temperatures in excess of 65° F. are usually required for the occurrence of the very short incubation periods.

Susceptibility and Resistance -

Adult salmon migrating up the Columbia River encounter massive numbers of bacteria, including flexibacters. By the time they reach the spawning grounds (1 to 3 months) they have developed high titers of protective antibody against flexibacters. If these "immunized" fish are forced to stay in warm water, above 68° F., serious losses still occur. The disease does not have as serious an effect on these fish as on unexposed fish, but it appears that these warm water temperatures so favor the columnaris organism that any resistance developed by the fish is eventually overcome.

Methods of Control

Prevention -

1. Provide cold water for salmonids. Pondfish also would benefit from cooler water if this can be provided without limiting productivity.
2. Maintain water supplies free of fish. Fish barriers should be built to prevent scrap fish from migrating above hatcheries using streams as water supplies.
3. Wherever columnaris is a problem, fish should not be handled or over-crowded during the warm water periods.
4. Maintain good water flows and remove dead and moribund fish from rearing facilities daily.
5. Re-use water only with extreme caution during the summer months.
-  6. Prophylactic treatment with external therapeutics have been reported beneficial. Furacin (nitrofurazone) at 5 - 10 ppm of active ingredients in distribution units and holding tanks has been used on bait fish. After 24 hours the treated water should be flushed out and a second 24-hour treatment started if necessary. Oxygen levels can be maintained with agitators during treatment.

Therapy -

-  1. The above furacin bath has also been used as a therapeutic treatment.
-  2. External infections, the most common form, have been effectively treated with Diquat at 8.4 to 16.8 ppm (2-4 ppm active cation) for 1 hour on 3 to 4 consecutive days. Copper sulfate at $\frac{1}{4}$ to 1 ppm has also been used in ponds for warm water species.
-  3. Internal infections, often a problem in advanced cases, have been treated with Terramycin at 3.0 gms per 100 pounds of fish for 10 days fed in the feed.

Peduncle Disease and Cold Water Disease

Introduction

A number of observations by different fish pathologists as well as recent laboratory tests have shown that both cold water disease and peduncle disease are caused by the same bacterium, Cytophaga psychrophila, a myxobacterium. Cold water disease is common in coho salmon and lake trout

sac fry and first feeding fingerlings. Peduncle disease generally attacks older salmon and trout. Workers in Oregon have observed that coho fingerlings which survived outbreaks of cold-water disease later suffered from peduncle disease and crooked spines. Close examination revealed myxobacteria in cysts along the spine in the area of the caudal peduncle. These cysts finally erupt to the surface developing into classical peduncle disease. Serological studies have shown that the two diseases are indeed related.

Cold water disease is characterized by an external infection of sac fry and young fingerlings. If it breaks out when the sac fry are within two weeks of "buttoning up" and are being reared in 40 - 50° F. water, the losses may exceed 50 percent of the population. If it breaks out after most of the fry swim up, losses can usually be held to 20 percent or less.

Signs

External -

1. In sac fry the thin epithelium covering the yolk is eaten away, spilling out the contents. This occurs in the incubation facilities and is first detected when bits of yolk plug the screens.
2. In swim-up fry and first feeding fingerlings, the most striking sign is erosion of the peduncle area and the eventual loss of tails.
3. Lesions start as pale white or bluish patches.
4. In older fish, lesions appear as scooped-out areas of flesh. If death does not intervene, the entire peduncle area erodes away, leaving only the exposed spinal column and a few fin rays of the tail.

Internal -

1. Internal signs are best determined through histological techniques; but, careful microscopic examination will reveal a systemic infection with C. psychrophila.

Epizootiology

Range -

The geographical range of cold-water disease is unknown but probably duplicates much of the range of susceptible salmonids. Peduncle disease occurs widely in a number of salmonid species.

Season of Occurrence -

Cold-water disease occurs in the spring before water temperatures rise above 50° F. while fry are swimming-up and beginning to feed. Peduncle disease occurs in the summer in fingerlings and yearlings.

Susceptibility and Resistance -

Coho salmon fry and fingerlings are primary targets for cold water disease. Sockeye and fall chinook fingerlings, affected by coagulated yolk disease, are also quite susceptible. Dr. John Schachte in New York reports evidence supporting the vertical transmission of cold water disease as an egg contaminant. Resistance to cold water disease develops with age. Little is known about the susceptibility and resistance of fish to Peduncle disease.

Methods of Control

Prevention -

-  1. Disinfect all salmonid eggs both after water hardening and upon receipt after shipping. Betadine at the rate of 6,667 ppm for 10 minutes has been used effectively. Eggs must be supplied with adequate oxygen during treatment.
2. Workers in Washington State have found that serious outbreaks of cold water disease may be prevented by lowering the water flow rate in incubation facilities to a quantity that will maintain a minimum of 4 to 5 ppm of dissolved oxygen at the outfall of the unit.

Therapy -

-  1. Sulfamethazine has been effective when fed to first feeding fry at the rate of 10 to 20 grams of drug per 100 pounds of fish per day in the starter diet for 10 - 20 days. A 5 gram dosage has been used for fingerlings.
-  2. On coho salmon fry and fingerlings, Hyamine 1622 at 2 ppm of active ingredient for 1 hour, has been administered 2 - 4 days per week along with sulfa therapy.

Bacterial Kidney Disease (BKD)

Introduction

Bacterial kidney disease is an infection of salmonid fishes which is characterized by its chronic, yet insidious nature. The disease is slow to become evident but once an outbreak begins it is difficult to control and virtually impossible to cure completely. The disease is caused by a small Gram-positive diplo-bacillus which has been identified as a new genus and species and is named Renibacterium salmoninarum.

The course of the disease is that of a chronic bacteremia. Once the bacteria enter the fish with infected food or are contracted from other infected fish on the hatchery or in the water supply, the organisms multiply slowly in the blood stream. Foci of infection

gradually develop in the kidney and in other organs such as the liver, spleen and heart. White pus and cellular debris collects in the blisters and ulcers that develop in these organs until the lesions are easily seen. The lesions developing in the posterior kidney are usually the easiest to find. They may reach a centimeter or more in diameter and extend into the musculature resulting in externally visible blebs under the skin. Once the disease has reached the stage in which gross lesions are apparent, therapeutic treatment has little effect. The most that can be hoped for is that drug therapy will possibly cure lightly infected or newly infected fish. Lack of an effective control for kidney disease is the basis for concern about this disease problem.

Signs

External -

1. The body of fish may be speckled with red spots (petechiae) giving them a "buckshot" appearance.
2. "Popeye" may be common, but is not unique to bacterial kidney disease alone.
3. Hemorrhagic areas at the base of the fins are common.
4. Large, sub-cutaneous welts are often seen on brook trout while tiny unbroken pimple-like lesions or small craters may be seen on the sides of rainbow trout and chinook salmon. In rainbow trout broodstock small, hard skin nodules have been found to contain kidney disease bacteria when no organisms could be found elsewhere in the fish.
5. Kidney disease bacteria are seldom detected in fish smaller than about 3 inches long or less than 6 months of age.

Internal -

1. Infected fish often have a swollen kidney which has a corrugated or "rope-like" appearance.
2. White boils of various sizes, filled with a creamy white fluid may be found chiefly in the posterior kidney and also in other organs such as the liver, spleen and heart.
3. The body cavity often will be filled with fluid.
4. Inflammation of the internal organs is not usually severe.

Diagnosis

The diagnosis of kidney disease depends upon the identification of the causative organism on stained slides prepared from moribund or dead fish. No other organism infecting salmonids could be confused with the kidney disease organism. It is a small (about 1 micron in length),

Gram-positive, non-motile diplo-bacillus that occurs in tremendous numbers in victim fish. The bacteria most often occur in pairs and are linked in a very shallow "V" configuration. If Gram-stained slides are prepared from crushed kidney material from fish which died of kidney disease, and examined under oil immersion on the microscope, there will be no difficulty in diagnosing the disease. Fluorescent antibody techniques have been developed which reliably confirm the presence of BKD.

Epizootiology

Range -

The geographical range of kidney disease follows the geographical range of salmonid fishes. The disease has not been found to naturally infect fish other than salmonids. Enzootic foci of kidney disease are numerous in the Pacific Northwest, around the Great Lakes and along the Appalachians from Georgia to Maine. Those hatcheries utilizing stream water supplies encounter kidney disease most frequently because native fish above the hatchery carry the infection continually.

Season of Occurrence

The largest number of cases of kidney disease occur in the months of March, April and May. It appears likely that initial infection occurs some months earlier and breaks out as the water temperature rises in the spring. In both trout and salmon, an increase in loss rate among yearlings accompanies warming springtime temperatures.

Incubation Period -

The incubation period of kidney disease varies with the water temperature. At temperatures above 52° F., initial losses occur some 30 to 35 days following exposure. At temperatures between 45 and 55° F., the incubation period increases to 60 to 90 days.

Susceptibility and Resistance -

Kidney disease has never been found to infect fish other than salmonids. Of the salmonids, salmon are somewhat more susceptible than trout. The disease is detected most frequently in spring chinook, coho and sockeye salmon since these species are more commonly reared to yearling age. In the Pacific Northwest a cross between the pink salmon and the chum salmon and called a "Chumpy" was found to be extremely susceptible. In trout culture, kidney disease attacks brook trout most severely, brown trout moderately and rainbow trout least severely.

Male broodstock trout have been observed to be grossly infected, whereas females in the population were apparently less severely involved. Whether the males become infected and suffer long-term chronic infections

while females become more acutely infected and die thereby leaving the males appearing more involved is not known. The carrier state among adult salmonids is common and is likely a main source of infection. Infected salmon viscera and adult-salmon carcasses were fed to fingerling salmon for many years virtually guaranteeing the perpetuation of kidney disease. No link with the diet remains with present processed feeds. Recent studies have shown that BKD is transmitted from adult carriers to their progeny through the egg. Infected eggs cannot be freed of BKD organisms by iodophor disinfection. This severely complicates disease containment and explains why the disease occurs as it does.

Methods of Control

Prevention -

1. Do not introduce eggs taken from BKD carriers into hatcheries free of the disease.

 2. To interrupt vertical transmission salmon culturists have injected 11.0 mg/kg of erythromycin phosphate, sub-cutaneously, into adult salmon as soon as they arrive at holding ponds. Additional dosages have been given at 30 day intervals until 30 days prior to spawning.

 3. When brood fish cannot be injected, salmonid eggs have been water-hardened in a 2 ppm solution of active erythromycin phosphate for up to 1 hour.

4. Maintain water supplies and supply systems free of fish.

5. Avoid the transfer of infected or exposed fish from hatchery to hatchery.

6. Maintain only those broodstocks free of bacterial kidney disease.

7. Always disinfect rearing facilities between uses.

Therapy -

1. Sulfamerazine fed at the rate of 5 grams of drug per 100 pounds of fish for 10 to 15 days may check the disease. Once mortalities are brought to near normal a continuous dosage of 2 grams of sulfamerazine per 100 pounds of fish should be fed until the fish are planted.

 2. Sulfamethazine used as described above has also been effective.

 3. Erythromycin thiocyanate fed at 4.5 grams per 100 pounds of fish for 21 days has also been used effectively. Disease recurrence should be anticipated. This drug is more expensive than the sulfa drugs.

4. No evidence of drug resistance has been detected to date with low-level sulfa prophylaxis for kidney disease.

PARASITIC DISEASE

The culture of fish in large numbers in crowded populations, when compared with natural population densities, provides conditions that are often conducive to the development of serious parasitic invasions. The simple close proximity of one fish, or host, to another is important to the success of some parasites. Another problem centers around the intentional enrichment of waters to stimulate zooplankton production for small fish feed. Such enrichment often provides favorable environmental conditions for the parasites of fish.

Many parasitic infestations can be controlled successfully by chemical treatment. Others, because of their nature, are virtually impossible to control and therefore present serious problems to the fish culturist who can only try to prevent them.

Effective control depends largely upon early detection and prompt corrective action whenever practical. The purpose of this section is to provide information helpful in the detection, identification, and control of parasite problems which are commonly encountered in fish hatcheries.

Key To The Major Groups of Parasites

- 1 - Microscopic (except Ichthyophthirius); single celled animals. PROTOZOA
 - Usually visible to the naked eye (some monogenetic trematodes, metacercariae, larval nematodes and cestodes are not, but they are multicellular) 2
- 2 - Body flat, more or less leaf-like; however, some monogenetics are nearly cylindrical 3
 - Body not flat 6
- 3 - Body segmented 4
 - Body not segmented 5
- 4 - In alimentary tract; worm has no digestive tract; possesses a scolex (head), neck, and series of proglottids (larvae not segmented, see #5) most CESTODA
 - External parasites; worm with digestive tract; possess anterior and posterior suckers (leeches) HIRUDINEA
- 5 - External parasites; posterior organ of attachment (haptor) well developed with chitinous anchors and hooks TREMATODA: MONOGENA
 - Internal parasites; usually an oral and ventral sucker, adults with eggs; in alimentary tract, bladders, occasionally elsewhere; metacercaria (larvae) in various organs (skin, muscle, mesenteries, etc.) with no eggs TREMATODA: DIGENEA

- In alimentary tract; no true suckers or segmentation some CESTODA
- Internal parasites; in mesenteries muscles, etc.; scolex with four suckers or two bothria (slit-like grooves); usually small but some can be seen with no magnification; calcareous corpuscles always present Larval CESTODA
- 6 - Body cylindrical; internal parasites 7
- Body not cylindrical (Lernaea nearly so); external parasites 8
- 7 - Anterior with hook-bearing proboscis; no digestive tract . . ACANTHOCEPHALA
- Anterior without proboscis; with digestive tract; adult female with eggs, male with testes; larvae without eggs or testes NEMATODA
- 8 - Body segmented with external appendages (difficult to see on Lernaea); on gills (except Lernaea on body and Argulus free-swimming on body); usually seen with naked eye COPEPODA
- Cyst-like structure on gills and/or fins containing delicate bivalve shell; clamping hooks or larva can be seen Mussel GLOCHIDIA

Some North American Fish Parasites
Listed by Host Location

1. EGGS

Fungi: Saprolegnia and relatives.

Protozoan: Carchesium (walleye and trout eggs; only record). Epistylis reported from catfish eggs.

2. SKIN AND FIN SURFACES

Protozoa: All protozoa and fungi of #1 above and #4 below.

Trematoda: Gyrodactylus, but usually not other monogenetics; metacercariae of many species.

Copepoda: Argulus, Lernaea, occasionally Salmincola.

3. CAUDAL FIN

Philometra carassii between fin rays of Carassius auratus.

4. GILLS

Fungi: Dermocystidium (salmon and trout).

Protozoa: Ambiphrya (Scyphidia), Amphileptus, Bodomonas, Chilodonella, Colponema, Cyclochaeta, Epistylis, Ichtyobodo (Costia), Ichthyophthirius, Microsporidea, Myxosporidea, Oodinium, Trichodina and Trichophrya on gill arches and filaments.

Trematoda: Gyrodactylus and other monogenetics; certain metacercariae.

Copepoda: Achtheres, Ergasilus, Lepeoptheirus, Lernaeocera, Salmincola.

5. BLOOD

Protozoa: Trypanosoma and Cryptobia free, Babesiosoma, Dactylosoma, and Haemogregerina in red cells, rarely Kudoa (Myxosporidea)

Trematoda: Sanguinicola in blood vessels, including gill vessels, also some migrating larval forms.

Nematoda: Philometra obturans in gill vessels of pike, Russia.

6. BRANCHIAL CAVITY

Trematoda: Syncoelium.

7. MOUTH

Trematoda: Some monogenetic, Leuceruthrus.

8. ESOPHAGUS

Trematoda: Azygia, Cotylaspis, Derogenes, Proterometra.

Arthropoda: Larval mites.

9. STOMACH

Protozoa: Schizameoba in fingerling trout.

Trematoda: Allocreadium, Aponurus, Azygia, Caecicola, Centrovarium, Derogenes, Genolinea, Hemiurus, Leuceruthrus

Nematoda: Haplonema

10. INTESTINE AND PYLORIC CAECA

Protozoa: Hexamita trophozoites and cysts in lumen, Schizamoeba cysts in fingerling trout, Eimeria in goldfish and trout.

Trematoda: Adults of many species.

Cestoda: Adults of many species.

Nematoda: Adults of many species.

Acanthocephala: Adults of many species.

11. BODY CAVITY: MESENTERIES, LIVER, SPLEEN

Protozoa: Many Myxosporidea species; rarely Microsporidea.

Trematoda: Many metacercarial species, including white grub (Posthodiplostomum); adult Paurorhynchus (bucephalid), adult Acetodextra in catfish.

Cestoda: Larval Diphyllbothrium, Haplobothrium, Ligula, Proteocephalus, Schistocephalus, Triaenophorus.

Acanthocephala: Larvae of Echinorhynchus salmonis, Leptorhynchoides thecatum, Pomphorhynchus bulbocolli.

Nematoda: Adult Philonema, many larval species.

12. GALL BLADDER

Protozoa: Myxosporidea, (Hexamita? Europe).

Trematoda: Crepidostomum cooperi, C. farionis, Derogenes, Plagioporus sinitsini.

Cestoda: Eubothrium salvelini.

Nematoda: Rhabdochona.

13. HEPATIC BILE DUCTS

Trematoda: Phyllodistomum spp.

14. KIDNEY

Fungi: Ichthyophonus hoferi.

Protozoa: Myxosporidea in renal tubules and also cysts.

Trematoda: Metacercaria of Posthodiplostomum minimum, P. centrarchi, and Nanophyetus salmincola. Adult Phyllodistomum in renal tubules and ureters.

15. URINARY BLADDER

Protozoa: Myxosporidea, Vauchomia (trichodinid).

Trematoda: Acolopenteron (monogenetic), Phyllodistomum.

16. OVARY

Protozoa: Wardia (Myxosporidea).

Trematoda: Adult Acetodextra in catfish. Nematobothrium in Ictiobus.

Nematoda: Philonema.

17. TESTES

18. SWIM BLADDER

Trematoda: Acetodextra

Nematoda: Cystidicola

19. HEAD SINUSES

Nematoda: Philometra

20. CARTILAGE

Protozoa: Myxosoma cerebralis (Myxosporidea) in trout, M. cartilaginis in bluegills, M. scleropercae in cartilaginous sclera or eye of perch, M. hoffmani in sclera of eye of Pimephales promelas, Henneguya brachyura from fin ray of Notropis, H. schizura from sclera of Esox lucius, Henneguya sp. from branchial arch of Pomoxis.

21. EYES

Protozoa: Rarely Myxosporidea in eye capsule.

Trematoda: Diplostomulum in lens and vitreous humor.

22. NERVOUS SYSTEM

Protozoa: Myxosoma cerebralis (Myxosporidea), effect on central nervous system, but parasites in cartilage, Myxobolus neurobius in spinal and peripheral nerves of salmonids in Europe, Myxobolus encephalica in brain of carp in Europe.

Trematoda: Diplostomulum, Ornithodiplostomum, Parastictodora, and Euhaplorchis metacercariae on brain; Psilostomum metacercariae in lateral line canal.

23. MUSCLE AND BODY CONNECTIVE TISSUE

Protozoa: Many Myxosporidea species, Sarcocystis.

Trematoda: Many metacercarial species, including yellow grub (Clinostomum) and black spot (Neascus).

Cestoda: Larval Triaenophorus, Diphyllbothrium.

Acanthocephala: Larval forms.

Fungal Diseases

Introduction

Fungal diseases are encountered by all freshwater fishes at one time or another. In hatcheries certain fungi are particularly troublesome. Filamentous fungi of the genus Saprolegnia are the most common and are well known for their infestations on fish eggs and fish. Also grouped with the fungi for convenience is the non-filamentous Ichthyophonus, which is probably a member of the class Fungi Imperfecti.

Ichthyophonus

Ichthyophonus has been reported as the cause of losses of both fresh and salt water fishes. In hatcheries it has caused serious, crippling lordosis and scoliosis in yearling rainbow trout. It is transmitted from infected non-game fish (carp from Utah Lake, Utah in one instance) to hatchery fish by feeding the infected trash fish as trout food for the hatchery trout. Viscera removed from infected fish in the packing process and then fed back to the production fish has also been shown to be a means of transmitting the disease. There is no control once infection has started but the disease may be easily avoided.

Saprolegnia

Saprolegnia is commonly encountered on fish eggs and on fish. It is recognized as a white, cottony growth consisting of a mass (mycelium) of non-septate filaments (hyphae) each of which is about 20 microns in diameter.

Saprolegnia establishes itself on dead organic material; dead eggs being an excellent growth medium. Fungi growing on dead eggs soon extend to involve and smother adjacent healthy, live eggs. Prompt picking of dead eggs will control Saprolegnia but this is difficult to do while the eggs are in the "tender stage" and egg picking is time-consuming.

☞ Good control has been reported by treating eggs on a "pre-need" basis with malachite green at 2.2 ppm for one hour, one or more times per week. Formalin at 1667 ppm (1:600) for 15 minutes is also highly successful. In no case should hatched fry be exposed to the egg treatment solutions.

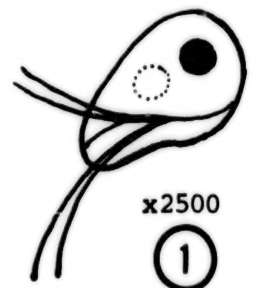
☞ Saprolegnia infections of fish often indicate injury or lesions from systemic infections. In the latter case it may be very difficult to successfully control the fungus. Malachite green flushes at 1 ppm, 3 percent salt baths for about 10 minutes, or standing treatments with malachite green at 0.3 ppm to 3 ppm have been successful. These treatments have been reported as toxic to some species of fish. To determine toxicity these treatments have been tested on a few fish before treating large numbers.

External Protozoa

Flagellates

Ichtyobodo (Costia)

Identification - Ichtyobodo is a small protozoan which attacks the skin and/or gills of fish and, because of its small size may be overlooked in an examination. Ichtyobodo is free-swimming, moves by means of long flagella, and is about 5 x 12 microns in size (Fig. 1).



Signs - An early sign of an Ichtyobodo infestation is a drop in appetite of the fish and a general listlessness. "Flashing" may be evident if the skin is infected but this sign normally is not seen if only the gills are infected. Heavily infected fish may develop a bluish slime over the entire body, however, fish less than 3 or 4 months old usually will die before this develops.

Control - Maintain water supplies free of fish. If Ichtyobodo is a frequent problem, treat with formalin at 167 ppm to 250 ppm for one hour on a "pre-need" basis when only light infections are present. Signs of Ichtyobodo may be easily mistaken for bacterial gill disease. Do not use formalin if fish have bacterial gill disease or serious post-treatment losses may be encountered.

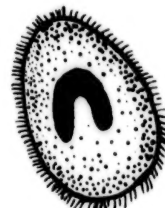
Ciliates

Ichthyophthirius -

Identification - "Ich" is a large ciliated protozoan that is exclusively a parasite of fish. It is a common parasite of warm-water fishes but also can be a serious parasite of salmonids. "Ich" is the only protozoan parasite that can be seen by the naked eye and when full grown may be as large as 0.8 mm and appear as gray-white lumps much like grains of salt. The most striking feature of the parasite is the large horse-shoe shaped nucleus that is visible in live specimens under the microscope (low power objectives) and positively identifies the organism. (Fig. 2.)

"Ich" are found under the epithelium of the skin, fins, and gills. When mature, the adult parasites drop off the host and attach to the bottom or sides of the pond. Once encysted there, they reproduce by fission and within one to several days, depending upon temperature, each adult gives rise to up to 2,000 young, ciliated "tomites".

The tomites burst from the cysts and must find a fish within about 24 hours or die. On contact with the fish the tomites penetrate the skin and begin to grow into adults. At optimal temperatures of 70 to 75° F. the life cycle may take as few as 3 to 4 days. The cycle requires 2 weeks at 60° F., more than 5 weeks at 50° F. and at lower temperatures may take months to complete.



x50

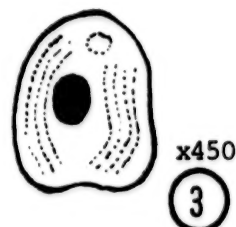
2

Signs - "Ich" is known as "salt and pepper" disease by aquarists because of the gray-white specks appearing on the skin. In severe outbreaks, losses may precede the appearance of the mature parasites on the fish. Young fish may exhibit considerable "flashing" or skipping off the bottom and often show erratic spurts of activity, jumping out of the water and thrashing about as they are irritated by the tomitte penetrations.

Control - Successful treatment of "Ich" depends upon the elimination of the stages of the parasite that are free in the water. The tomites and the adult parasite leaving the fish are, therefore, the target of therapeutic efforts. Repeated treatments at daily intervals in warm water and less frequently in colder water is required. Formalin at 167 ppm to 250 ppm for 1 hour or at 25 ppm in earthen ponds is recommended. Malachite green at 0.25 ppm for 1 hour or at 0.1 ppm in ponds has also been successful and has been used in a combined treatment with the formalin. Early and repeated treatment is essential to good control.

Chilodonella -

Identification - Chilodonella is a small, oval, colorless protozoan, 50 to 70 microns long, which may be found in vast numbers on the skin, fins, and gills of goldfish, other warm-water species and salmonids. Under high magnification, faint bands of cilia can be seen over much of the organism (Fig. 3.). It has an optimum temperature of 40 to 50° F. which makes it particularly troublesome on warm-water species during cold weather.

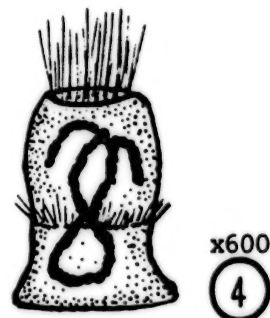


Signs - Heavily infected fish are listless, do not feed actively and may "flash".

Control - Formalin at 167 ppm to 250 ppm for 1 hour or at 25 ppm in ponds easily controls Chilodonella. This treatment should not be used in ponds during hot weather.

Ambiphrya (Scyphidia)

Identification - Ambiphrya are small, urn-shaped organisms with a ring of cilia at their "top" end and a band of cilia around their "middles" (Fig. 4.). These protozoans can be found in large numbers on the gills, skin and fins of salmonids and warm-water fish species. They virtually smother the gills by sheer numbers of parasites present on the gill surface.



Signs - Heavy gill infestations may present the signs of bacterial gill disease. Heavy skin infections are not common but may cause listlessness and "flashing".

Control - Formalin at 167 to 250 ppm for 1 hour easily controls Ambiphrya. In ponds, formalin at 25 ppm usually rids fish of these parasites.

Epistylis -

Identification - Epistylis is somewhat similar in appearance to Ambiphrya but smaller and grows in clumps at the ends of non-contractile stalks (Fig. 5.). Under the microscope they appear much like a cluster of bluebells growing on a stalk that is attached to the fish by a disc. They are usually found on the skin but may be found also on the gills.

Signs - "Flashing" during the late morning and late evening hours is one of the first signs of infestations. These parasites evidently bother the fish with little apparent tissue damage. Epistylis should be removed if it is causing severe "flashing" as the fish will suffer skin abrasion if allowed to continue, thereby affording an opening for fungus or bacterial infection.

Control - Epistylis is easily removed by a 167 ppm formalin treatment for 1 hour if the water temperature is 55° F. or above. Below 45° F. Epistylis may not be removed by formalin at 250 ppm for one hour unless the treatment is repeated once or twice. Epistylis may attack egg masses of warm-water species. Formalin at 1,667 ppm for 15 minutes should be tested for control.

Trichodina -

Identification - Trichodinids are saucer-shaped protozoans with rows of cilia primarily around the margin of the body as it is normally viewed on the microscope (Fig. 6.). These protozoans live on the skin, fins, and gills of fish and, when abundant, cause considerable irritation to these tissues.

Signs - In severe cases almost continual "flashing" may be observed. Salmon yearlings, if left untreated, "develop a rather tattered appearance". Secondary bacterial infection may also develop in untreated cases.

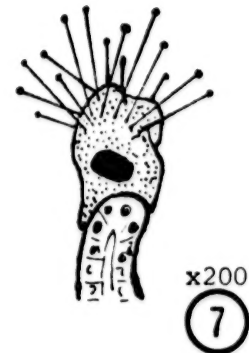
Control - At hatcheries where Trichodina is a chronic problem, treatment should be planned on a "pre-need basis". Formalin at 167 ppm to 250 ppm for 1 hour is usually successful. Where salmonids are sensitive to formalin, a 2 to 4 ppm treatment of Diquat for 1 hour has been reported as effective. Formalin at 25 ppm in ponds is usually successful.



Suctorina

Trichophrya -

Identification - Trichophrya grossly resembles Ambiphyra in appearance but instead of cilia around the "top" it has small tentacles giving it the appearance of a small pincushion (Fig. 7.). These parasites are found on the gills and may merely be along for the "ride", feeding on aquatic organisms. When abundant, they interfere with respiration and cause proliferation of the gill tissue.



Signs - The signs presented by an infestation of Trichophrya could easily be mistaken for bacterial gill disease. Fish will appear listless and have a poor appetite.

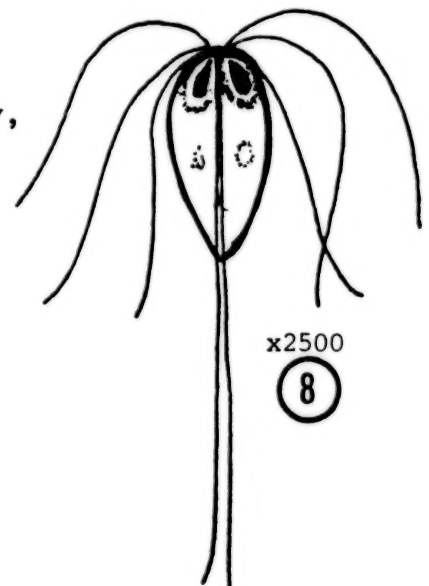
Control - Formalin at 167 to 250 ppm for 1 hour or at 25 ppm in ponds has been successful. Stubborn cases may require more than one treatment, if so, repeat in 10 days.

Internal Protozoa

Flagellates

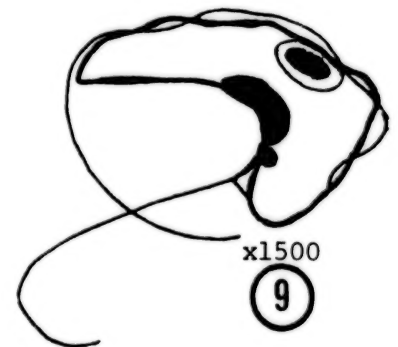
Hexamita -

Hexamita, formerly known as Octomitus, is a tiny, highly motile protozoan bearing eight flagella, that is found in the intestines of fish (Fig. 8.). It may become abundant in fish fed meat diets and may cause irritation of the lining of the gut. With the advent of processed diets the incidence of Hexamita has greatly declined. Therapy is not recommended unless Hexamita are abundant. For treatment, epsom salts (magnesium sulfate) fed at the rate of 3 per cent of the diet for 2 to 3 days has been effective.



Cryptobia -

Cryptobia is a microscopic flagellate found in the bloodstream of fish (Fig. 9.). In severe cases these protozoans become so abundant in the bloodstream that they may outnumber the red blood cells causing severe anemia. These parasites are transmitted from fish to fish by leeches and are found in a wide variety of fish species including salmonids in many parts of the world. There is no known control other than measures to eliminate leeches.



Ceratomyxa shasta -

Identification - Ceratomyxa shasta is a microscopic protozoan parasite of salmonids which causes severe losses of rainbow and steelhead trout and chinook and coho salmon in the western United States (Fig. 10). Heavy exposure of salmonid fingerlings can result in 100 percent mortality. The disease can be contracted by adult salmon upon entering infected fresh water and heavy pre-spawning losses of spring chinooks can occur. The spores of Ceratomyxa shasta are elongate and are found consistently in the intestine and gall bladder of infected fish. Lakes or ponds are apparently critical to the development of the infective stage of this parasite a fact that is of great importance to the prevention of the disease in hatcheries. At about 50°F. the life cycle can be completed in 20 to 30 days. Little is known of the intermediate stages of the life cycle.



x2000

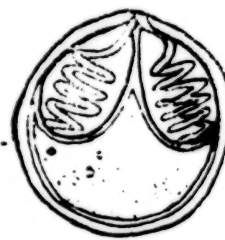
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Signs - In fingerlings a gross swelling occurs in the area of the vent. Abdomens may be distended with fluid and caseous material can be found around the viscera. Mortalities may exceed 10 percent per week in some cases. Some strains of fish may be more susceptible than others.

Control - There is no known treatment. This parasite should be avoided at all costs. Water supplies, known to be infected, should not be used for fish culture unless the inflow is effectively sterilized. There should be no transfers of young fish or adults from infected areas to non-infected areas.

Myxosoma cerebralis (Whirling Disease) -

Identification - Whirling disease, a mad tail-chasing activity, is caused by Myxosoma cerebralis. (Fig. 11). Trout with twisted spines and deformed heads result. Gill cartilage may be the best tissue in which to find the tiny lemon-drop shaped spores with two polar capsules. At 50 F. spores can be found about four months after initial exposure. The sporoplasm does not contain an iodophilous vacuole as contained by members of the genus Myxobolus which look nearly identical to Myxosoma cerebralis.



x3500

11

Signs - Severe infections in young rainbow trout fingerlings often cause death before spores develop. Survivors may develop a "black tail" condition due to the loss of control of chromatophores because of damage to the spine and associated nerves. At two to eight months of age rainbow or brook trout may appear normal but, when startled or at feeding time, may break into frenzied tail chasing. Whirling subsides as the fish grow older and twisted spines and deformed heads are the major signs of whirling disease thereafter. Identification of the spores is required for confirmation.

Control - As with Ceratomyxa, there is no known therapy. Control lies with preventing the introduction of infected or exposed fish, mud, or water. There is hope that infected facilities can be disinfected, but much work remains.

Henneguya -

Identification - Henneguya spores are distinctive long, twin-tailed spores. Henneguya exilis frequently has been reported from channel catfish but a similar species of Henneguya has caused catastrophic losses of fingerlings. H. exilis (Fig. 12.) is found in cysts in the blood capillaries while the more dangerous form is found in the inter-lamellar spaces of the gills. The inter-lamellar form develops cysts that may fill as much as 90 per cent of the space between the gill lamellae causing suffocation. It can be distinguished from H. exilis by the presence of polar filaments which are coiled, cork-screw fashion, at the tips while the tips of the polar filaments of H. exilis are straight.

Signs- In channel catfish, visible white to pinkish cysts are found in the gills, frequently in huge numbers. Fish may appear to suffer from an oxygen deficiency even in water with 8 ppm dissolved oxygen. Losses may exceed 95 percent in fingerlings less than two weeks old. Mature cysts are filled with typical spores.

Control - As with other Myxosporidean infections, prevention is the best control measure as no treatment is possible. The disease has been spread from hatchery to hatchery with shipments of infected fingerlings. The confirmation of the inter-lamellar form in a catfish population warrants destruction of the infected fish and disinfection of the rearing facilities involved.

Microsporidea

Plistophora -

Several species of Plistophora have been found to infect hatchery fish. As the name of the class Microsporidea indicates these are exceedingly small protozoans. Plistophora are about the size of large bacteria, being 3 to 6 microns long and bean-shaped (Fig. 13.). Severe infections have been reported in the gills of rainbow trout and in the ovaries of golden shiners. In the golden shiner the parasite infests up to about half of the ovary and significantly reduces the fecundity of broodstock populations. The only known control is prevention. Fish or eggs should not be transferred from infected hatcheries to uninfected hatcheries. Broodstocks known to be infected should be phased out and the rearing ponds disinfected.



x1500
12



x6000
13

TrematodesMonogenetic TrematodesIntroduction -

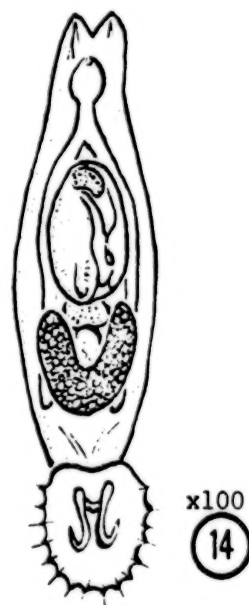
The monogenetic trematodes of hatchery fish are capable of completing their life cycle on the fish without involving other animal hosts. Young worms are virtually identical in appearance to their parents. The majority are too small to be seen by the naked eye but some species may reach 5 mm in length. The organ of attachment, the "haptor", which is posterior, is the best aid to the identification of different genera and species. Nearly always there are 12 to 16 marginal hooklets around the haptor and 2 to 4 large anchor hooks in the center.

Species of the family Gyrodactylidae are generally found on the body and fins but are sometimes also found on the gills. These parasites move around freely. The members of this family give birth to live young, similar in appearance to the adults. They have no eye spots.

Species of the family Dactylogyridae commonly are found on the gills of fish. Dactylogyrids lay eggs into the water. Most species have eye spots, 1 to 2 pairs of anchor hooks and 14 marginal hooklets. Dactylogyrids are common on warm-water fish species while Gyrodactylids are common on trout and on warm-water fish species.

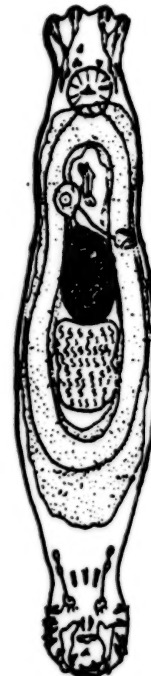
Gyrodactylus -

Gyrodactylus can be identified by the developing embryo inside the adult along with the lack of eye spots. The haptor has 1 large pair of anchor hooks and 16 marginal hooklets (Fig. 14). Under optimum conditions these worms can be seen by the naked eye. These worms are so common on trout that it is unusual to examine fish and not find them. They may occur in large numbers and cause skin irritation making treatment necessary to remove them. Fish with large numbers of Gyrodactylus may appear listless, have frayed fins and "flash" frequently. These parasites are easily removed by treating the fish with formalin at 167 to 250 ppm for 1 hour or at 25 ppm in ponds. Potassium permanganate at 2 to 3 ppm for 1 hour should be tested as an alternate treatment for formalin-sensitive trout.



Dactylogyrus -

Dactylogyrus is but one of a number of genera of Dactylogurids found on warm-water fish. These worms are particularly serious parasites of Cyprinids. Dactylogyrus, primarily a small gill parasite, can be identified by the presence of four eye spots, one pair of relatively small anchor hooks, and 16 marginal hooklets, of which two are small (Fig. 15.). No embryo will be found inside, as these worms lay eggs. These parasites, when numerous, can cause serious damage to the gills. Signs of the disease, could be mistaken for those caused by an oxygen deficiency or other gill infections. Formalin at 25 ppm in ponds or at 167 to 250 ppm in raceways for 1 hour has been effective.

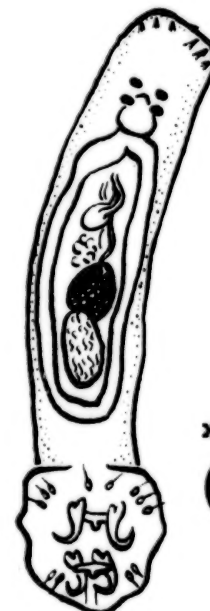


x200

15

Cleidodiscus -

Cleidodiscus is common on the gills of catfish and a wide variety of other warm-water fish species. It is similar in appearance to Dactylogyrus in that it has eye spots. A distinguishing characteristic, however, is the presence of 4 large anchor hooks (Fig. 16.). These worms, like Dactylogyrus, lay eggs and unlaidd eggs can be seen within the adult worm. Cleidodiscus is found only on the gills. When numerous it causes respiratory problems by severely damaging gill tissue. The signs, therefore, are those of gill damage and may be similar to those seen when oxygen is low. Formalin at 25 ppm in ponds or at 167 to 250 ppm in tanks and raceways for 1 hour usually removes these parasites.



x200

16

Digenetic Trematodes

Introduction -

The digenetic trematodes of hatchery fish require one or more hosts other than the fish to complete their life cycle. These parasites can be divided into two major groups; (1) those that live in fish in their adult stage, producing eggs which leave the fish to continue the life cycle, and (2) those that penetrate the skin of the fish and live in the fish as metacercariae, usually encysted in the tissue, until the fish is eaten by the final host.

Sanguinicola (Blood Fluke) -

Blood flukes are adult trematodes that live in the gill arterioles in the gill arches of salmonids and other species of fish. These tiny worms lay non-operculate eggs which become trapped in the capillary beds of the gills and other organs where they develop into miracidia which have a characteristic dark eye-spot. When fully developed the ciliated miracidia burst from the gill to be eaten by an operculate snail which is the only intermediate host in the life cycle. Cercaria emerge from the snail and seek fish to complete the chain.

Control of blood flukes is difficult at best but depends upon preventing cercarial penetrations of the fish through continual treatment of infected water supplies to kill the cercaria or through the eradication of the intermediate host snails. In most cases, however, blood flukes are not the cause of serious losses of fish. It is conceivable that large numbers of miracidia leaving the gill at one time could cause loss of blood and damage to the gills. Eggs and developing miracidia also interfere with the circulation of blood in the gill capillaries and in the capillary beds of the kidney and liver.

Nanophyetus salmincola (Salmon Poisoning Fluke) -

These parasites are present in salmonids as metacercariae awaiting the eventuality that an infected fish will be eaten by carnivorous mammals such as dogs, bears, foxes, and raccoons. Infested mammals shed eggs into the water which hatch as free-swimming miricidia. The miricidia bore into a snail, Oxytrema silicula, where they multiply. The cercariae leave the snail and, upon contact with a fish, bore in and encyst as metacercariae in nearly any tissue. Large numbers of metacercariae have a debilitating effect upon young salmon. Massive penetrations by cercariae may kill young salmon, but the same number penetrating over a prolonged period has little apparent affect.

No treatment has been devised to completely rid streams of snails or rid fish of encysted metacercariae. Pre-treatment of infected water supplies may be an effective way to prevent infection.

A brief note on "salmon poisoning". Throughout the life cycle of the fluke, it is, itself, parasitized by a rickettsian. It is this rickettsian, rather than the worm, which causes "salmon poisoning disease" in dogs. The family pet should not be allowed to eat raw salmon or even lap up the blood of such fish where these parasites are enzootic.

Neascus (Black-spot Disease) -

Many species of freshwater fish have been found to be infested with small black spots in the skin which are slightly raised. If the spots are numerous they are more easily noticed and in severe cases more than 50 per cent of the body and fins of some fish may be covered with black spots. These spots result from an infestation of young flukes of the larval genus Neascus. The adult worm is a parasite of a fish-eating bird. Droppings from the bird carry eggs into the water where they hatch into miracidia which attack a snail. In the snail a complicated developmental process takes place and after a month or so, an enormous number of cercariae emerge. The cercariae, upon encountering a susceptible fish, burrow into the skin and encyst as metacercaria. The fish, in turn, surrounds this cyst with a black-pigmented tissue giving rise to the black spots. There is no known control and the only recognizable problem is the appearance of infected fish.

Diplostomulum spathaceum (Eye Fluke) -

The eye fluke is a member of a group of metacercaria that are parasites of a wide variety of fish. One of these species (D. spathaceum) is found in the lens of the eye and two other species are found in the vitreous chamber of the eye. D. spathaceum is the species most frequently encountered in fish hatcheries. The final host is the sea gull which releases mature eggs into the water with its droppings. After hatching, the miracidia seek snails of the genus Lymnaea. Cercariae emerge from the snail and penetrate the skin of the fish. A few successfully find their way to the eye and encyst in the lens. Serious blinding of large numbers of hatchery fish can occur if conditions are suitable. There is no known treatment for infected fish.

Posthodiplostomum minimum -

Metacercariae of Posthodiplostomum minimum are common parasites in the viscera of hatchery centrarchids. They are seldom a problem. Under conditions favorable to the parasite, however, they occasionally have been observed to be the likely cause of severe losses of striped bass fingerlings in North Carolina. Heavily infected fish had popeye caused by the large number of encysted metacercariae behind the eye, and a grossly swollen area below the eye. Snail control was suggested as a means of controlling the problem in North Carolina. Some workers have suggested the use of snail-eating sunfish for control of these intermediate hosts. Redear sunfish have been found to reduce P. minimum infestations through the control of snails. This method of controlling snails to ultimately control digenetic trematodes should be tested wherever redear sunfish could live together with production species.

There are many species of Strigeid trematodes; therefore, several species of fish-eating birds and snails may be involved in individual cases.

Clinostomum marginatum (Yellow Grub) -

Yellow grubs are the metacercaria of the digenetic trematode Clinostomum marginatum. The metacercaria are 2 to 4 mm in length and are often found in the musculature of infected fish. The discovery of the metacercaria in sport-caught fish is often disgusting to fishermen.

Infestations are seldom extremely heavy but individual fish may be highly parasitized. The life cycle involves the great blue heron or other fish-eating birds. The worm matures in the mouth of the bird. Eggs of the parasite are shed into the water as the bird feeds. The eggs hatch after a few hours in the water and the miracidia penetrate certain species of snails. After development, free-swimming cercariae leave the snail and penetrate beneath the scales of a variety of fresh-water fishes where they become encysted in the loose connective tissue under the skin.

Cestodes

Introduction

Tapeworms are long, flattened, noodle-like, segmented worms which taper toward the head end. Close examination of this end of the worm will reveal features that provide clues to the parasite's identification. The oldest segments are at the tail of the worm. Each segment, or proglottid, contains a complete set of both male and female reproductive organs and may contain large numbers of eggs.

Two life-cycle stages are found in fish. The adult worms are found in the intestine and pyloric caecae while the plerocercoids (larval tapeworms) are found in the viscera or musculature. The first larval stage, the proceroid, is found in the hemocoel of aquatic copepods, amphipods, and isopods.

Only heavy infestations of mature tapeworms in the intestines are believed to be detrimental. Large numbers of adult Corallobothrium may retard the growth of catfish. Plerocercoids, however, are much more damaging. These larval forms migrate through the body cavity and internal organs causing adhesions to develop. Severe adhesions in adult bass females, caused by the plerocercoids of Proteocephalus, render the females virtually incapable of normal egg production and spawning. In small fish the migrating plerocercoids may actually kill the fish by penetrating a vital organ.

Corallobothrium -

Corallobothrium could properly be called the catfish tapeworm as the adult worm is seldom found in other than Ictalurid fishes. Heavy infestations impair the growth of catfish and may become a hatchery production problem. The eggs pass from the catfish with the feces and larval proceroids can later be found in the copepod Cyclops. The next larval stage, the plerocercoids, are found in shiners.

Corallobothrium can be identified by the fleshy folds of tissue on the head end that cover 4 suckers (Fig. 17.). The individual segments in the mature tail-end of the worm are usually quite distinct.



x40

17

Adult tapeworms have been removed from fish by feeding di-N-butyl tin oxide at the rate of 0.3 per cent of the ration for 3 days. Bayer 2353, forcefed in capsules at the rate of 226 milligrams per pound of fish should be tested for tapeworm removal in broodstocks. There is no known treatment for the control of tapeworm plerocercoids in the viscera or muscle.

Proteocephalus (Bass Tapeworm) -

Several species of Proteocephalus may be found in a wide variety of fresh water fish species. This tapeworm has been given the common name of the bass tapeworm as Proteocephalus ambloplitis is commonly found in the adult stage in the intestine of both largemouth and smallmouth black bass. The plerocercoids are usually found in small fish but may occur in adult fish where they may be a serious problem in fish culture. The adult may be identified by the presence of 4 clearly discernable suckers on the head of the worm (Fig. 18.). Treatments for Proteocephalus are the same as for Corallobothrium.



x40

18

Diphyllbothrium -

Only plerocercoids of Diphyllbothrium are found in fish. The adult tapeworm is found in the intestines of birds, cats, dogs, bears or man depending upon the species of worm. Migrating plerocercoids can cause extensive damage in small fish. The larvae are non-segmented but are wrinkled and may give the impression of being segmented (Fig. 19.). The head end is compressed laterally and has two elongate shallow grooves (bothria). The larvae may be found loose in the viscera or burrowed into the musculature. When abundant, parasitized fish may be listless and swim near the surface where they may be easily caught by hand.



x5

19

Plerocercoids of D. latum, one of several species found in hatchery fish, are found in northern pike and walleyes. The plerocercoids are found in the muscles, viscera and connective tissue. Human beings become infected with the adult worms by eating raw or undercooked fish. There is no treatment for the control of plerocercoids in fish. If human infestation is suspected, obtain medical assistance promptly.

Nematodes

Nematodes are long, thin, round, un-segmented worms with a smooth outer surface. Most fish nematodes, in the adult stage, can be found in the intestinal tract. Adult Philonema and Philometra may be found in the tissues of fish, however. Larval nematodes of fish may be found in almost any tissue or organ, but are most common in the mesenteries, liver, and musculature.

The first intermediate host in the nematode life cycle is always an invertebrate such as a copepod or an insect nymph. These intermediate hosts are eaten by fish as they forage. If the fish is infested by a larval stage, the final host must eat the fish before the adults can develop in the intestine of fish-eating fish, birds, and mammals.

Certain nematodes such as Capillaria may cause inflammation of the lining of the intestine in warm-water hatchery fish, but most nematodes produce little pathology by their presence. Philonema can cause severe visceral adhesions in salmonids. There is no known control for nematode infestations.

Acanthocephalans

Acanthocephalans are well-named as "thorny-headed worms". Their thorny, hook-bearing, retractable proboscis has most of the characteristics needed to identify the species of these worms found in fish.

Although serious losses due to Acanthocephalans are seldom encountered in fish culture, heavy infestations may cause inflammation in the intestine by the numerous thorny heads of the worm buried in the intestinal wall.

The Acanthocephalan life cycle is straightforward. Eggs shed by the adult worm residing in the intestine of the fish are eaten by copepods, isopods, amphipods or ostracods. In these crustacea, the first larval stage, or acanthor, migrates into the body cavity of the crustacean and develops into the second larval stage, the acanthella. Usually there is no second intermediate host and the final host becomes infected by eating the infected crustaceans, completing the life cycle. Obviously this could easily take place in a hatchery pond. There is no known treatment. Fish cultural practices calling for the drying of ponds for a portion of the season may aid in preventing serious infestations.

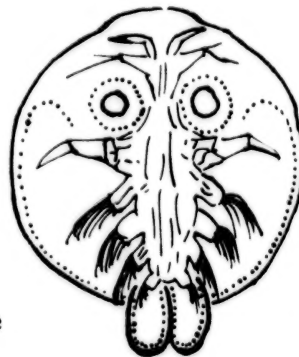
CopepodsIntroduction

The vast majority of the copepods in fresh and salt water are an important part of the diet of fish. Certain species, however, are parasitic on fish and the infested sites on the fish may become ulcerated and provide an access for secondary infection by fungi and bacteria. Crowded hatchery rearing units provide ideal conditions for infestations by copepods because of the dense fish populations and rich environmental conditions. Under most hatchery conditions, however, serious losses of fish are seldom caused by parasitic copepods alone. The stocking of copepod-infested fish in streams has been shown to be a means of infecting wild fish. Infested fish are considered "wormy" by sportsmen and the parasites may inhibit growth and productivity of fish populations.

Argulus (Fish Lice) -

Argulus sp. have been given the common name fish lice as they have the ability to creep about over the surface of the fish. On first glance they look like a scale but on closer examination are seen to be saucer-shaped and flattened against the side of the fish. They have jointed legs and two large sucking discs for attachment which may give them the appearance of having large eyes (Fig. 20.).

Argulids penetrate the skin of the host fish, inject a cytolytic substance, and feed on blood. Argulus has been shown to transmit infectious dropsy of carp. If these organisms become abundant, even large fish may be killed. Dylox at 0.25 ppm has been used for the treatment of Argulus in ponds. Complete drying of rearing units will kill free living eggs, larvae and adult.

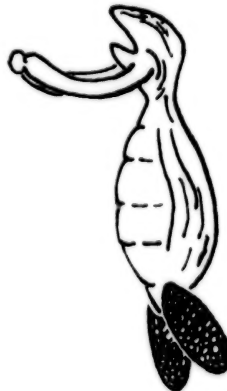


x10

20

Actheres ambloplitis -

Catfish commonly are infected with A. ambloplitis in their gills and are thought to be "wormy". The light colored body of the parasite stands out in sharp contrast to the deep-red gill filaments. The legs of this copepod have become modified through successive molts to form an attachment organ to hold the parasite in place on the gills of the fish. Egg sacs extend from the posterior end of the body of the female (Fig. 21.). The adult female is the form usually found in fish. Heavy infestations by larval forms may kill fish by severely damaging the gills. There is no known treatment for the control of Actheres.



x10

21

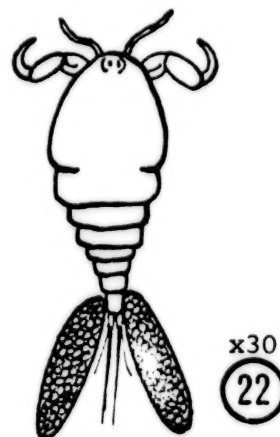
Salmincola -

Grossly similar in appearance to Actheres (Fig. 21.), Salmincola is a gill, mouth and fin parasite of salmonids. Losses are seldom attributable to Salmincola, but fishermen complain about "wormy" or "grubby" fish when they catch fish which are infested.

Fish infested with Salmincola are virtually impossible to cure on a production basis. These parasites may be eliminated from a hatchery if the water supply can be controlled. Eggs and larval stages cannot stand drying and the larvae must find a fish host within a day or two or die. If all parasitized fish can be eliminated completely from the water supply and the infected facilities allowed to dry for three or four days, complete eradication of Salmincola is possible. If rearing units cannot be completely dried, the fish should all be removed and the remaining water treated with 200 ppm chlorine and left free of fish for three or more days. The maintenance of water supplies free of resident fish is essential to good parasitic copepod control.

Ergasilus -

Ergasilus is a small (1.5 to 2.5 mm), cyclops-like gill parasite which is found attached to the gills of a wide variety of fish species by means of clasper-like claws (Fig. 22.). Impregnated females are the only Ergasilids found on fish and they produce eggs at intervals of 3 to 12 days depending upon the temperature and species of parasite. Up to 1 million eggs may be produced during the 1-year life span of the female. Eggs hatch in 2 to 4 days and another 10 to 70 days is required for the copepod to reach sexual maturity. No known treatment has been developed for Ergasilus but a related genus, Pseudergasilus has been successfully controlled with a combination of 0.5 ppm copper sulfate and 0.2 ppm ferric sulfate for 6 to 9 days. This should be tested locally before large-scale application.

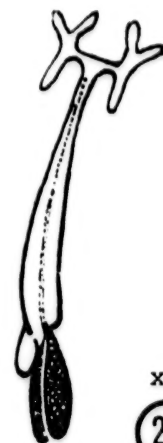


Lernaea (Anchor Worm) -

Lernaea are most commonly found on warm water fish species. But one species of Lernaea, L. cyprinacea, lacks host specificity and even attacks frogs and salamanders. Heavy infestations have caused massive mortalities in carp and goldfish populations. The parasite destroys scales and causes a sore at the point of penetration. The main damage is loss of blood and exposure to secondary infection by fungi, bacteria and possibly viruses.

Lernaea is a long (5 to 22 mm) slender copepod which, when attached, gives the appearance of a soft stock with two egg sacs attached. Actually, the head-end is buried in the flesh. This end has large, leathery horns which aid in identification of the parasite (Fig. 23.).

These parasites have been successfully controlled with the gamma isomer of benzene hexachloride. However, this compound is not biodegradable and should not be used. Potassium permanganate, at 25 ppm in tanks and raceways for 90 minutes or prolonged treatment at 2 ppm in ponds, has been successful for treating carp. The 25 ppm level may be toxic to some fish species and the use of this compound for Lernaea control should be tested further. Dylox at 0.25 ppm has been effective. Usually four or more treatments, at weekly intervals, have been required to eliminate the parasite because of the continuing hatching and development of young.



VIRAL DISEASES

There are several viral diseases of fish in the United States and Europe which pose a serious threat to populations of both wild and domestic stocks of fish. The viruses responsible are sub-microscopic, absolute parasites which are completely dependent upon the living cells of the host for their reproduction and which cannot be grown outside of living cells on artificial media. Most viruses are so small that they may be passed through filters which retain bacteria and thus have been termed "filterable viruses". All known viruses are considered infective agents and they often have highly specific requirements for the type of host and for certain tissues within the host. The pathological damage they cause in host tissues or in cultured cells is often characteristic of the specific virus involved and provides useful diagnostic information. The disease syndromes that result fall into two major types: those characterized by an inflammatory response or tissue destruction, and those which are characterized by abnormal tissue growths or tumors.

Protection against viral infection is best provided by specific immunization of the host through vaccination with specially prepared viral material that does not result in disease but effectively stimulates the development of immunity. Unfortunately no immunizing vaccines are available for use in fish culture. Certain chemotherapeutics have been found effective against certain viruses of higher animals but again none have been shown to have reliable effect on fish viruses. Until

protective or curative treatments are developed, prevention of viral diseases is the key to their control. The prevention of viral infections depends upon our ability to detect virus-carrying fish populations and isolate them. Hatcheries which are known to be free of viral diseases are a special asset and must be aggressively protected from these diseases. Infected hatcheries should initiate programs to progressively eliminate the diseases or minimize their impact. The decision to control viral diseases is largely administrative. Well thought out and imaginative disease eradication plans can be implemented effectively without seriously impacting fisheries management programs.

Infectious Pancreatic Necrosis (IPN)

Introduction

Infectious pancreatic necrosis (IPN) is a salmonid virus disease which is widespread in North America and has been spread to Europe. IPN outbreaks have been reported in brook, rainbow, cutthroat, brown and lake trout. It has also been reported in Atlantic salmon and more recently in Pacific salmon. IPN virus is egg transmissible. This makes IPN especially dangerous as it quickly and easily can be shipped with eggs spawned from IPN carriers. The disease is the cause of high losses among young fingerlings but may cause few signs if the fish are over 6 inches in length when exposed. Many survivors develop into IPN carriers which may spread the disease to other fish.

Signs

External -

1. In young fingerlings, a sudden increase in the daily death rate, particularly among the larger and healthier-appearing fish, is often the first sign of an IPN outbreak.
2. Some of the affected fish will spiral or roll through the water as they swim, especially if they are touched or otherwise disturbed. This spiralling is along the long axis of the body and is not a "tail-chasing" activity.
3. Before death, moribund fish will often lay against the screen of the rearing unit at the outlet, too weak to swim against the current.
4. Moribund fish are often dark in color and may commonly display external and internal signs which are virtually indistinguishable from those caused by a systemic bacterial infection. Hemorrhage is often found at the base of the fins, popeye may be observed, and sick fish may have a "blotchy" pattern of external coloration.

Internal -

1. A mucus plug in the stomach and anterior intestine has been reported for brook trout fingerlings but this sign is variable and may not be found in all species.
2. Red specks of hemorrhage are common on the viscera and internal body wall.
3. The liver is often pale or blotchy, giving the suggestion that losses may be due to nutritional problems.
4. The digestive tract is usually empty.
5. Histologically, IPN virus is found to be highly specific for pancreatic tissue and there is massive necrosis of the acinar cells accompanied by pyknotic nuclei.

Diagnosis

Reliable diagnosis of IPN requires isolation of the virus in cell culture and identification through serum neutralization procedures. The RTG-2 cell line is the cell culture of choice and a poly-valent immune serum has recently been made available which greatly strengthens confirmation procedures. Any young trout that dies with suspected IPN signs and appears to be free of bacterial and parasitic infections should be subjected to virological examination.

IPN virus is the smallest known fish pathogen and measures about 65 nanometers in diameter. It is remarkably stable and retains some infectivity in physiological solutions even after an hour at 60° C. The virus is insensitive to ether and may be stored in 50 percent glycerin and conveniently mailed to the laboratory, but if any other known fish viruses are present they will not survive this treatment. The virus has been placed tentatively with the Reovirus group but further study may place the IPN virus in another group or in a group of its own.

Epizootiology

Range -

IPN follows the geographical distribution of trout but not all waters and not all hatcheries have been exposed. The ease of shipping infected eggs has much to do with the distribution of this disease. It is known to occur through most of North America and has been reported, since 1965, from several northern European countries.

Season of Occurrence -

The bulk of the cases of IPN occur in June and July. There is an elevated incidence rate during the first five months of the year due to the large populations of small fingerlings in production at that time. When warmer waters are encountered in the early summer outbreaks peak sharply. Relatively few cases are encountered from August through the end of the year.

Incubation Period -

The incubation period has been determined, experimentally, to be 6 days at 54° F. in brook trout fingerlings fed IPN virus. It is longer in rainbow trout; up to several weeks at 40° F. Conversely, higher water temperatures would accelerate the course of the infection.

Susceptibility and Resistance -

Vigorous, fast-growing fish may be more vulnerable to IPN than poorer quality fish. It is speculated that the healthy, rapidly-metabolizing tissues of the robust fish offers a high quality environment for virus replication. Of the trout species, rainbow and especially brook trout are considered to be the most susceptible to IPN. Losses of young brook trout often reach 90 to 100 percent.

Methods of Control

Prevention -

1. Inspect all hatchery fish populations at least annually for the presence of IPN.
2. Phase out all IPN-infected broodstocks and ship no eggs from such broodstocks to any hatcheries capable of operating without contracting the disease from their own water supply.
3. Ship no IPN-exposed fish to watersheds where IPN is not known to exist.
4. Do not transfer exposed fish or eggs to hatcheries which are free of the disease.
5. Under no circumstances should infected fish be stocked into lakes, reservoirs or streams which serve as water sources for hatcheries.

Therapy -

1. There is no known treatment for IPN once it has been contracted by the fish.
2. Strict sanitation may prevent the spread of IPN to fish in adjacent rearing units. The effluent from infected fish rearing units is usually highly infectious and all fish exposed to this water should be considered exposed and probably infected.

3. Hyamine 3500 at 1,000 ppm is an effective disinfectant for treating hatchery troughs, incubators and associated equipment. When this compound is used, thoroughly rinse facilities and equipment before use to flush away possibly toxic residues of the chemical. This is especially important for incubators and egg-handling equipment.

Viral Hemorrhagic Septicemia (VHS)

Introduction

Viral hemorrhagic septicemia (VHS) has not been found in North America but is a serious hatchery problem in most western European countries. Epizootics have been reported in brown trout but VHS is primarily a disease of rainbow trout. It attacks catchable or marketable trout with severity but is seldom a problem among young fingerlings or broodstocks. The disease is spread from fish to fish through the water and may not be egg transmissible. Fish culturists should guard against this eventuality, however.

Signs

External -

1. Acute form: found at the beginning of an outbreak and characterized by darkly colored fish, popeye on one side of the fish, and hemorrhage in the eyes and at the bases of the pectoral fins. Heavy losses are common.
2. Chronic form: found following the initial acute form and characterized by extremely dark colored fish, popeye on both sides of the fish, and severe anemia.
3. Nervous form: found near the end of an outbreak and characterized by frenzied activity rather than lethargy, fish swimming in circles, and loss of balance.

Internal -

1. Extensive hemorrhaging in the muscles is one of the most common internal signs. The fish have a badly "buckshot" appearance throughout the the normally clear muscle tissue.
2. Speckles of hemorrhage are also found throughout the visceral mass.
3. The kidney, early in an outbreak, is bright red and swollen. Later it becomes greyish and somewhat corrugated.
4. Red blood cell counts are frequently as low as 300,000 per cubic millimeter.
5. The digestive tract is usually empty and the pH of the stomach is 6 or 7 rather than the normal 1 or 2.

Diagnosis

Diagnosis of VHS depends upon the isolation and identification of the viral agent. The RTG-2 cell line is the cell culture of choice. The cultures should be young with a pH of the media at about 7.6 to 7.8. All cultures should be incubated at 15° C. Anti-IPN serum will have no neutralizing effect on VHS virus and the cytopathic effect (CPE) to look for in the cell cultures consists of small round holes in the cell sheet which are rimmed with rounded cells. The centers of these holes are clear of cellular debris.

VHS is caused by a bullet-shaped virus about 60 - 70 nanometers in diameter and 180 nanometers long. The virus is ether-sensitive and cannot be preserved in 50 per cent glycerin.

Epizootiology

Range -

VHS has been found in Europe quite extensively. It has not been diagnosed in North America. Outbreaks are most severe in cold water below 50° F. and the severity decreases as water temperatures rise into the 60's. Since this disease remains exotic to North America any cases, in which the symptoms of VHS are detected, should be reported to a fish disease laboratory for prompt investigation.

Season of Occurrence -

Late winter and early spring are the seasons in which the most serious outbreaks occur.

Incubation Period -

Deaths due to VHS, in natural infections, occur 7 to 12 days after exposure. At 60° F. fish die 10 to 15 days after intraperitoneal injection.

Susceptibility and Resistance -

VHS is primarily a disease of rainbow trout but outbreaks have occurred in brown trout populations. Experimentally brooks, browns and Atlantic salmon have been infected. Grayling and whitefish may also be susceptible.

Methods of Control

Prevention -

Preventive measures against VHS, in this country, consist largely of preventing the introduction of the virus through importation of infected eggs or fish. No salmonid eggs or fish may legally enter the United States unless they have been thoroughly inspected and found free of VHS.

Therapy -

As with other viral infections, there is no known treatment once VHS has been contracted by the fish.

Infectious Hematopoietic Necrosis (IHN)

Introduction

Infectious hematopoietic necrosis (IHN) is a viral disease of rainbow trout that is caused by a virus believed to be a member of a closely related group of viruses. The viruses causing IHN, Sacramento River chinook disease (SRCD) and sockeye salmon virus disease (SSVD) have been found to be serologically related. Because of this relationship and because IHN is more widely distributed, IHN will be discussed in some detail here. The interested fish culturist should consult Dr. S. F. Snieszko's "Symposium on Diseases of Fish and Shellfish" published by the American Fisheries Society in 1970 for details on the virus diseases of Pacific salmon.

Numerous outbreaks have occurred in salmon hatcheries but little is known of the extent of the threat this disease poses for trout hatcheries. The disease is known to be enzootic in rainbow broodstocks in certain locations in the Pacific Northwest. It was likely introduced with the untreated salmon viscera fed to these fish to produce high quality eggs. Eggs taken from these infected stocks have failed to transmit the disease unless they are contaminated with infected ovarian fluid after the eggs are well "eye-up" and nearing the time of hatching. Eggs taken from carriers are apparently unable to transmit the virus if they are reared in IHN-free water throughout the incubation period. These findings are only tentative at this time and fish culturists should not depend upon this observation as a means of preventing infection.

Signs

External -

1. IHN attacks very young fingerlings reared in water temperatures of 50°F. or less. Losses often exceed 90 percent.
2. Long opaque fecal casts trailing from the vent of the fry is quite common in outbreaks of IHN.
3. Victim fish are dark, lethargic and often have popeye.
4. Hemorrhagic areas above the vent, on the sides, is common as is hemorrhage at the base of the fins.
5. Gills are often pale and small hemorrhagic spots are sometimes found in the mouth.

6. In at least one case, about 5 percent of the survivors has scoliosis.

Internal -

1. Grossly, moribund fish infected with IHN, have pale livers, kidneys and spleens.
2. Ascitic fluid accumulations in the body cavity are common.
3. Some small hemorrhagic spots may be found throughout the visceral mass.
4. Inflamed intestines are common.

Diagnosis

Diagnosis of IHN requires isolation of the virus in cell culture and presumptive identification through a process of eliminating other viral agents as suspects. The CPE caused by IHN in RTG-2 or FHM cell cultures is characteristic but experience is required to be able to accurately distinguish it. Since the range of IHN overlaps the range of IPN, simultaneous infections by both agents are conceivable. In such an event, the PPN virus could be neutralized with specific anti-sera and the IHN would continue to cause CPE in treated cultures. IHN inoculated cell cultures should be incubated at 15° C. for 10 to 15 days and checked frequently for the development of grape-like clusters of cells surrounding lysed areas in the cell sheet.

IHN virus is a bullet-shaped virus which measures about 90 by 158 nanometers. The virus is not stable in ether and cannot be stored in 50 percent glycerin.

Epizootiology

Range -

Outbreaks of IHN which have occurred at hatcheries outside enzootic areas of the Pacific Northwest and British Columbia have been attributed to the shipment of contaminated eggs or fry from this area. The disease thus may have been established in some of those areas but largely is restricted to the Northwestern states.

Season of Occurrence -

Outbreaks have coincided with the trout fry and early fingerling season of April to June. IHN could occur at any time susceptible young fish are exposed.

Incubation Period -

The incubation period has been determined experimentally by adding 2 to 3 ml of infected cell culture medium to 400 ml of water containing 20 small fish exposing them for one hour. The fish showed fecal casts on the third or fourth day and started dying on the fourth or fifth day. After 14 days 60 to 90 percent of the fish were dead.

Susceptibility and Resistance -

An interesting relationship has been demonstrated between the sockeye salmon virus and the IHN virus. When rainbows are exposed to the sockeye virus only a few die. When sockeye fingerlings are exposed to IHN virus only a few die. When fingerlings are exposed to their own viruses, heavy losses occur. Rainbow trout and sockeye salmon are both susceptible to SRCD virus if exposed before they reach one month of age. After one month these species cannot be infected by SRCD virus by merely adding it to the water. Age of the fish at exposure has a big role in the impact of this group of viruses. Temperature also greatly affects the severity of outbreaks and a 59° F. rearing temperature has been employed at the Coleman National Fish Hatchery to prevent the outbreak of SRCD. Cold water temperatures (below 50° F.) favor IHN, a fact that is borne out by work in cell culture.

Methods of Control

Prevention -

1. Rainbow trout eggs should be obtained from inspected sources shown to be free of IHN.

-  2. IHN transmission, with eggs taken from carrier adults, has been effectively halted by soaking eyed eggs in an iodophor disinfectant solution, such as Betadine, that provides 100 ppm of iodine, at pH 6.0 or higher, for 10 minutes.

Therapy -

1. There is no known therapy for IHN unless one considers the use of warm water to control outbreaks in chinook salmon fingerlings a therapeutic measure. The question remains, however, as to whether the virus is only rendered dormant by the higher temperature and will become active again upon finding favorable conditions. Work on this problem is continuing in California. Warm water temperatures do not prevent IHN losses in rainbow trout or sockeye salmon fingerlings.

Channel Catfish Virus Disease

Introduction

Several outbreaks of channel catfish virus disease (CCVD) have been reported from the South Central states. At first the losses were blamed on bacterial infections but prompt antibiotic therapy failed to bring relief. Extensive virological investigation in 1968 proved these outbreaks were due to channel catfish virus. Since that time research has continued and several serious outbreaks have been reported. The threat to the booming catfish industry is significant.

Signs

External -

1. During warm weather when water temperatures reach the upper 70's, a sudden increase in moribund fish is usually the first indication of the disease.
2. Fish can be seen swimming abnormally and spiralling through the water as do trout infected with IPN.
3. Convulsions are common, after which the fish rest on the bottom.
4. Just before death the fish hang at the water surface in a tail-down position. This sign is most characteristic.
5. Hemorrhage may be found at the base of the fins and speckled over the abdomen. Gills may be hemorrhagic.

Internal -

1. Hemorrhagic areas are common in the musculature, liver, kidneys and spleen.
2. The stomach may be distended and may contain a yellowish mucoid material.
3. Popeye, caused by kidney failure, is common.
4. Edema is common.

Diagnosis

The diagnosis of channel catfish virus is often obscured by the presence of known bacterial fish pathogens capable of killing fish. Aeromonas hydrophila and columnaris may be present in moribund fish. Antibiotic therapy for these bacterial infections may clear them up but losses will continue as drugs such as Terramycin have no effect on the virus. Confirmation of channel catfish virus disease depends upon the isolation of the virus in cell culture. BB (brown bullhead) cells are the cells of choice. BB cells

should be inoculated with bacteria-free filtrates of ground viscera diluted no more than 100 fold. CPE develops in 3 to 7 days in BB cells incubated at 27° C. CPE is characterized by large rounded cells with vacuolated cytoplasms.

Epizootiology

Range -

Channel catfish virus disease has been confirmed in Alabama, Arkansas, Georgia, Kentucky, Mississippi and Texas and in other states where suitable environmental conditions occur.

Season of occurrence -

Confirmed outbreaks occurred during the warm water season of July, August and September. In three of the outbreaks, the disease was preceded by either handling stress or low dissolved oxygen levels but these conditions may have been coincidental. Field observations indicate that water temperatures above 70° F. are likely essential before outbreaks occur.

Incubation Period -

The incubation period of channel catfish virus disease is temperature dependent. Experimental infection at 86° F. was followed by symptoms 2 to 3 days later and the first deaths a few hours afterward. At 68° F. it took about 10 days for symptoms to develop and the first deaths did not occur until several days later. In natural outbreaks disease symptoms appeared 72 to 78 hours after exposure and 88 to 100 percent of the fish were dead within 6 days. Water temperatures ranged between 68 and 86° F.

Susceptibility and Resistance -

Channel catfish virus appears to be highly host specific both in the species of fish it will infect and the cell cultures it will attack. Channel catfish are the only known species of fish known to be susceptible. Other warm water species such as goldfish, bluegill, striped bass and redear sunfish are not susceptible. Only cell cultures derived from Ictalurid tissues have been found to be attacked by the virus severely enough to produce recognizable CPE. Fish to fish transmission is the likely means of spreading the disease. Extensive sampling of tissues from adults that had been exposed to channel catfish virus failed to detect the virus and no sampling methods have been established for reliably detecting virus carriers. Serological tests for neutralizing antibody in fish may be useful.

Methods of Control

Prevention -

1. Avoid hatchery to hatchery transfer of infected or exposed catfish.

2. If channel catfish virus disease is detected, the infected facilities should be isolated and the fish buried. The pond and all associated equipment should be disinfected.

Therapy -

1. There is no known therapy for viral infections.
2. Colder water may inhibit the progress of an infection and fish may be saved; however, all survivors should be considered to be carriers of the disease.

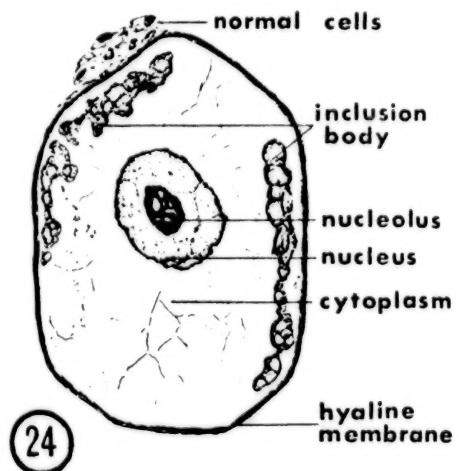
Lymphocystis

Lymphocystis is a virus-caused disease, which is common in walleyes and sometimes found in sunfish, that stimulates grossly abnormal growth of connective tissue cells. The disease is chronic and seldom causes the death of infected fish. Lymphocystis virus is the cause of benign, wart-like growths composed of enormous tissue cells which average about 100 microns in diameter; some may reach a millimeter in diameter. The lesions are white or pinkish and may become slightly hemorrhagic since they are easily irritated.

Careful examination of lesion material with a hand lens or a dissecting microscope will reveal the tremendous size difference between affected and unaffected cells. (Fig. 24.).

The virus causing this phenomenon is about 300 nanometers in diameter, resists drying and is glycerin sensitive.

There is no known control for lymphocystis other than removal of infected fish and preventing the infection..



MISCELLANEOUS AND MYSTERIOUS MALADIES

Nutritional Diseases

Cataract

Rainbow trout fed the USFWS trout diet during the late 1960's were frequently observed to develop opaque lens' in their eyes. This problem ranged from mild to severe but did not cause death. The cataract problem was believed to be due to a vitamin deficiency or due to the presence of

"white fish meal" in the formula. Newer formulations were designed to alleviate the problem while, simultaneously, research on the problem was conducted. It has since been found that cataracts often occurred in fish fed diets deficient in zinc. The cataract problem has declined greatly with the newer diet formulas utilizing herring meal rather than white fish meal but occasional outbreaks are seen as diet formulas change.

Nutritional Gill Disease

Nutritional gill disease has long been attributed to pantothenic acid deficiencies in the diet of salmonids. The disease is characterized by the display of disease symptoms similar to those caused by bacterial gill disease. Moderate to severe thickening of the epithelium occurs on the gill lamellae and clubbing of gill filaments soon follows. Gill filaments fuse together in advanced cases.

Nutritional gill disease will not usually be encountered if fresh food is being fed. Dry pelleted feeds which have been stored for long periods or exposed to high summertime storage temperatures usually can be incriminated.

Fish should be treated as if they have bacterial gill disease on a "pre-need basis" as the gills are highly susceptible to infection. Additional fresh water or aeration should be provided. Fresh diet should be obtained. Care should be taken not to over feed as the sick fish will not have much appetite at first. Recovery will be gradual but should not require more than a month under most conditions. When the fish have regained their strength they may be handled for thinning populations into additional rearing units if necessary.

Other dietary deficiencies caused by improper formulation or prolonged storage may lead to other vitamin deficiency diseases such as blue slime (biotin deficiency) or others.

Environmental Problems

Gas Bubble Disease

At various times fish culturists are faced with the problem of supersaturation of the water by nitrogen gas. Nitrogen is inert and as such cannot be metabolized and can build up in the blood of fish to the point at which bubbles of nitrogen form in the blood and tissues. This accumulation of gas can cause popeye, gas-filled blisters under the skin and bubbles in the fins and body cavity. Severely affected fry are sometimes so full of gas they can no longer stay down in the water and float helplessly at the surface upside down.

Various conditions cause supersaturation of hatchery waters. Natural waters from springs are sometimes supersaturated with nitrogen after the spring thaw because of the over-winter accumulation of nitrogen gas produced by natural breakdown of nitrates and nitrites. Well water may be easily supersaturated if air is sucked into the well casing because of congestion of the screen or a hole in the system above the water level. Aeration of supersaturated water may be helpful but the success of aeration in removing nitrogen is largely dependent upon the energy expended in the aeration process.

Ammonia

Ammonia is a waste product of metabolism and the amount released into a unit of water is related to the amount of metabolism going on in that unit of water. Ammonia may occur naturally in some ground waters. Ammonia is excreted through the gills of fish and fish need healthy gills to be able to do this efficiently. Excessive ammonia causes proliferation of the gill tissue much like that caused in bacterial gill disease. The affect of ammonia on fish depends upon the amount of free ammonia there is dissolved in the water. This is regulated by pH to a large extent. Roughly speaking, if the pH is on the acid side (less than 7.0) ammonia toxicity will seldom be encountered under normal fish cultural conditions provided the fish are the sole source of the ammonia. As the pH goes up and the waters become more alkaline, ammonia becomes more of a problem. When the pH of the water is below 8.0 total ammonia levels up to 0.5 ppm are seldom toxic. This 0.5 ppm figure is a good "rule of thumb" maximum for fish culture.

Oxygen deficiency

Terrestrial plants and animals live in an environment which provides oxygen at the relatively constant rate of 20.9 percent of the air respired. Fish and other aquatics are faced with variable oxygen levels some of which may reach supersaturation and some of which reach zero. The point at which fish culturists become concerned varies with the requirements of the fish. Oxygen levels of 6 ppm or less at the outfall of raceways or ponds in which salmonids are reared are usually considered low. To correct this condition more fresh water should be added; aerators can be used; and, most importantly, the density of the fish population can be reduced.

In warm water fish culture there are often few options and the problem can be complex. On bright sunny days phytoplankton blooms produce far more oxygen than they consume. On cloudy days such a bloom may produce oxygen sufficient to sustain itself but at night the phytoplankton must draw upon the dissolved oxygen in the water in direct competition with the fish. In late summer, when blooms are mature and fish population heavy, this competition for oxygen may become acute and total depletion of the oxygen may occur causing massive fish kills overnight.

The initial stages of fish kills caused by oxygen depletion can be recognized by characteristic symptoms. The larger fish may die first as small fish can crowd close to the surface to obtain oxygen. This "piping" at the surface is an important sign of trouble. Most fish kills occur late at night and are most severe just before daylight. The water color at this time has changed to a dark color and may have foul odor. By this time it is too late to save many fish.

To prevent such fish kills, fertilization must be carefully regulated so as not to encourage massive blooms. If a bloom appears to be getting too heavy, treatment with copper sulfate at 0.5 ppm is often highly successful although it may reduce the zooplankton population somewhat. Close observations of pond conditions and prompt action to limit the growth of phytoplankton blooms will be well worth the effort. Potassium permanganate at 1 to 2 pounds per acre may be used as an emergency treatment in ponds suffering from the onset of an oxygen deficiency problem.

Mysterious Maladies

Sunkburn

Occasionally salmonids, especially brown trout, develop dorsal skin lesions. It is characterized by a peeling or sloughing of the outer layer of skin along the dorsal surface. This first appears as a darkening of the skin between the head and the dorsal fin. When this layer sloughs off, the corium layer is exposed giving the area a white appearance. Further sloughing may lead to death. Dietary vitamin deficiencies have been thought to be a cause but crowded rearing conditions in concrete raceways should be considered as another. For relief, the fish should be thinned to additional raceways or to ponds.

Soreback

Rainbow trout reared in crowded concrete raceways in 55 to 60° F. water often have been affected by extreme dorsal fin erosion which progresses into the musculature in the vicinity of the dorsal fin. The lesions occur specifically at this site on all affected fish but the degree of severity may vary. Detailed experimentation has revealed that the lesions are due to "nipping" and that population density and insufficient feed are significant factors. Growth rates are usually rapid in 55 to 60° F. water and often the providing of extra food will minimize the problem. Soreback is seldom a problem in earthen rearing units.

Coagulated Yolk Disease (White-Spot Disease)

Millions of chinook salmon fry have been lost to coagulated yolk disease. A variety of extrinsic factors may precipitate the condition. Rough "shocking" of eggs at too-early a stage of development, incubation temperatures above 54 or 55° F., excessive use of malachite green on eggs and the presence of heavy metals in the water have all been incriminated as possible causes. The disease attacks young fry at first feeding and affected fish appear "blimpy" in that considerable yolk material has not been utilized. Close examination of the fish reveals bits and pieces of

cream-colored coagulated yolk material in the yolk sac. As the disease progresses the fins become rigid and are fully extended. White caseous material often coats the fins. Death soon follows. There is no known treatment for fish afflicted with coagulated yolk disease and little can be effectively done to prevent it until more is known about the etiology. Discontinuation of the use of malachite green for the control of fungus on eggs may be associated with a reduction in the incidence of coagulated yolk disease in recent years. Genetic susceptibility should be fully studied.

Soft-Egg Disease

Occasional batches of eggs will be found which are abnormally soft to the touch and sticky in texture. Affected eggs tend to stick together in clumps thereby predisposing them to fungus. The cause of soft-egg disease is believed, by some, to be due to bacteria which destroy the permeability of the shell which permits water to enter the eggs. It is rarely serious. Considerable success in controlling soft-egg disease has been had through treating the eggs with acriflavin at 500 ppm for 25 minutes

TREATMENT OF FISH DISEASES

Introduction

A complete rearing season seldom passes in which fish do not require treatment for one disease condition or another. It is not unreasonable to consider every treatment a dangerous undertaking and the fish culturist must take every possible precaution to avoid disastrous results. All drugs and chemicals used to control infectious organisms are toxic to fish and success is had only by treading the fine line between safety and disaster. Any calculation used should be double checked before going ahead; if possible, by a second person. In fish culture, unlike the physician or the veterinarian who treat patients on an individual basis under carefully controlled conditions, fish populations are treated en masse with great trust that the law of averages will work in our favor.

Two general methods of treatment are usually employed: (1) a therapeutic is incorporated in the feed of the fish, or (2) a therapeutic is added directly to the water in the rearing unit.

Therapeutics Added to the Feed

Two useful drugs have been cleared by the FDA for use in treating systemic bacterial infections in hatchery fish. These are Terramycin, an antibiotic, and sulfamerazine, a sulfonamide. There are other drugs which have not yet been cleared for use but are identified here in the event that they are cleared for future use.

Sulfonamides

Sulfamerazine is effective for treating infections caused by both Gram-negative and Gram-positive bacteria. In the body the action of the drug is basically bacteriostatic and the defensive mechanisms of the fish are what actually kill the bacteria. Tests have shown, however, that sensitivity test results are a very good indicator of the value of the sulfonamides for therapeutic purposes. Another sulfonamide,  sulfamethazine (Sulmet), is closely related to sulfamerazine in action, and has been used by West Coast workers for the treatment of bacterial infections in salmon.

Sulfonamide toxicity may occur when high dosages (10 grams per 100 pounds of fish per day or more) are fed to sensitive fish. If high dosages are required, a combination of two sulfa drugs totalling the desired high rate should be fed. Toxicity usually results from the formation of complexed sulfa crystals in the renal tubules when the urine is acidic. The signs of drug toxicity include abnormal bloating of the stomach with water (dropsy) and a chronic susceptibility of the gills to fungus (in salmon). Popeye, swollen kidneys and death are seen in acute cases. With the diets presently available, consumption of any added drugs is usually good. With the possible exception of bacterial hemorrhagic septicemia caused by A. hydrophila or P. fluorescens, high sulfa levels are seldom required.

Dosage rate: Treatment - Sulfonamides incorporated in processed diets are fed at the rate of 5 grams per 100 pounds of fish per day for 10 days. This therapy should produce a blood level of the drug of 9 to 12 mg per 100 ml of blood.

 Dosage rate: Prophylaxis - While it is seldom a good practice to feed low levels of antibacterial drugs, history has shown the effectiveness of a continual 2 gram, per 100 pounds of fish, level of sulfamethazine for controlling losses to bacterial kidney disease.

Terramycin (Oxytetracycline)

Terramycin is often dramatically effective against furunculosis, redmouth and other bacterial diseases caused by Gram-negative rods. It is ineffective against kidney disease. The drug is usually available in the form of TM-50 or TM-50D, the latter being a formulation with a water-dispersible carrier which can be used in the manufacture of starter diets or moist pellets which require very fine particles for processing. Terramycin, TM-50 and TM-50D are trade names of the Charles Pfizer Company for their oxytetracycline products. TM-50 and TM-50D contain 50 grams of oxytetracycline hydrochloride per pound of product. In other words, each 9 grams of TM-50 or TM-50D contains 1 gram of Terramycin.

Terramycin has been most useful in treating bacterial infections which have developed a resistance to sulfa. Drug resistant strains of bacteria, resistant to Terramycin have been encountered, however.

Dosage rate: Treatment - Terramycin fed at the rate of 3.0 grams per 100 pounds of fish per day for 10 days is usually effective. Terramycin often stimulates the appetite of trout but salmon may find it distasteful.

Dosage rate: Prophylaxis - Terramycin should not be fed on a prophylactic basis. Terramycin-resistant bacterial strains may develop which render the drug useless.

Furazolidone (Furox 50)

Furazolidone is one of a large number of related drugs in the nitrofuran group. The name Furox 50 is the trade name given a product of the Norwich Pharmacal Company for a formulation containing 11 percent furazolidone. This drug has provided good results in the treatment of furunculosis and redmouth in trout and against other Gram-negative rods in other species (especially in cases where drug resistance has been developed against the sulfonamides and Terramycin). Nitrofurans are suspected of being cancer-causing chemicals.

Toxicity of Furox 50 is no problem. Reports of toxicity of Furazolidone to brown trout in the late 1950's have largely been discounted as possibly caused by other things. When properly administered, no toxicity problems have been encountered in recent years.

 Dosage rate: Treatment - Furazolidone has been effective when mixed in the diet at the rate of 2.5 grams of pure drug per 100 pounds of fish for 3 days followed by a 20-day course of a 1.0 gram level per 100 pounds of fish. (NOTE: Furazolidone breaks down in wet (meat) diets. This drug has been fed in a pelleted feed. If a wet diet must be fed, it should be mixed fresh for each feeding.)

Dosage rate: Prophylaxis - Furazolidone should not be fed on a prophylactic basis.

Therapeutics Added to the Water

Three basic methods are commonly used to treat fish with chemicals added to the water. The first, and perhaps the most reliable method for raceways and hatchery tanks and troughs, is the standing bath treatment. The second, which can be used in nearly any situation where fish are raised in running water, is the constant flow treatment. The third is the prolonged treatment which is usually used for treating fish in large earthen ponds.

Standing Bath Treatment

At trout, salmon and catfish hatcheries where large numbers of fish are reared in troughs, tanks raceways and rectangular concrete ponds, good use of the standing bath treatment can be made. For this treatment the water is drawn down to a pre-determined level, the water supply is shut off, and the outlet is sealed against leakage for the duration of the treatment. The chemical is then added.

The treatment period is usually prescribed for 1 hour. Under no circumstances should this period be exceeded irrespective of how well the fish act. If needed, several short baths at daily intervals are far superior in every way to one extended treatment. Should signs of distress appear among the fish during the hour of treatment do not hesitate to terminate treatment for the day and try again the next day.

Standing bath treatments are accomplished by weighing or measuring the correct amount of the compound to be used, dissolving or diluting it in as large a quantity of water as practical, and mixing the solution with the pond water which has been adjusted to the correct treatment level. The solution should be mixed uniformly with the pond water to avoid "hot spots". This can be done by using aerators or long-handled pond brushes.

Special precaution must always be taken not to lower the water level too low for the weight of fish in the rearing unit. Also, the treatments recommended may not have a large safety factor; therefore, in no event should these amounts be exceeded. It does not hold that if a little is good, a lot is better.

Formalin - Formalin is probably the most widely used therapeutic agent in fish culture. It is the common name for a solution containing 37 grams of formaldehyde in 100 grams of water. Methyl alcohol or calcium salts may be added to prevent changes from occurring in the formalin. Formalin is corrosive and may turn rusty if stored in drums with scratched linings. Neither the rust nor the stabilizing chemicals added to formalin have an effect on formalin for treatment purposes. Old formalin or formalin exposed to cold temperatures may have a white precipitate in it. This is para-formaldehyde which is more toxic than formalin and, if prevalent, it should not be used.

Formalin is effective against the vast majority of external fish parasites with the possible exception of Saprolegnial fungus infections. Formalin is correctly used in troughs, tanks, raceways and rectangular concrete ponds at 167 to 250 ppm for 1 hour. Temperature of the water is extremely important. Salmonids are somewhat sensitive to higher levels of formalin above 45 to 50° F. As a rule of thumb use the 167 ppm concentration for 1 hour if the temperature is over 50° F. and repeat the treatment two or three times on succeeding days if the parasites have not been removed. In earthen ponds which cannot be flushed out following treatment, use 25 ppm of formalin and allow it to naturally dissipate. If rearing ponds are heavily loaded with either fish or phytoplankton, try a treatment of 15 ppm of formalin and repeat if necessary at 5 to 10 day intervals.

Formalin at 1667 ppm is effective against fungus which attacks eggs in incubation. The treatments should be of 15 minutes duration and may be repeated as often as is necessary to prevent fungal growth.

Fish treated with formalin may suffer a delayed mortality five to six hours after treatment. Rainbow trout yearlings are notorious for this problem, especially if they have never been treated with formalin before. Experience has shown, however, that if rainbow trout have been treated with formalin since they were young fingerlings, the older fish are seldom adversely affected. There are few concrete clues to the cause of formalin sensitivity and a few fish can be expected to be lost on the first treatment. Formalin is also highly toxic to humans. The strong odor and the eye irritation usually warn of its presence. Long term exposure to low level formalin fumes can be dangerous to human health and no one has ever survived swallowing over 60 ml of formalin.

 Malachite Green - Malachite green has a long history in fish culture. It has been used extensively to control fungal growths on fish eggs and for the external treatment of fungus on adult salmon being held for spawning. Unfortunately malachite green has been found to cause birth defects in experimental rabbits and may also be a cause of cancer. Because of these findings, many fish culturists no longer use malachite green and a major research effort is underway to find suitable alternative chemicals.

During its heyday, two basic approaches were used in the application of malachite green for the control of fungus on incubating eggs. The first was a constant flow drip method in which a stock solution of malachite green was metered into the incubation units at a rate that brought the inflowing water to 2.2 ppm (1:450,000) malachite green for 1 hour. The other method was the "California flush method". This technique involved preparing a stock solution of 42.5 grams of malachite green in 1 gallon of water. The inflowing water to the incubation unit was adjusted to a measured 6 gallons per minute and 88 ml of the stock solution was added, all at once, at the inflow point. After the dye cleared from the unit, the water flow was returned to the desired level. If a lesser or greater water inflow rate was necessary during the treatment, the amount of the stock solution added was adjusted accordingly. This method was successfully used where large number of eggs were being incubated.

Treatment of eggs for the control of fungus should only be carried out as needed. It should not be performed on a routine, scheduled basis because any agent capable of halting fungal growth is a toxic compound that could cause damage to developing embryos or lead to conditions such as coagulated yolk disease after hatching. In no case should hatched fry be exposed to anti-fungal egg treatment dosages.

External fungal infections are a serious problem in adult salmon being held for spawning. Other fish species also suffer fungus infections, but these are usually secondary to other conditions. A standing bath of 1 ppm malachite green was used to treat these outbreaks. This treatment was also useful in controlling columnaris if detected early. At temperatures over 58° F. this treatment was repeated as often as 4 days per week. If fish are being handled, fungal patches and open sores have been swabbed with a 100 ppm solution of malachite green or with standard tincture of merthiolate as purchased from the drug store. The colored areas on the skin helped fish culturists to observe treatment progress.

 Hyamine 3500, Hyamine 1622 and Roccal - These compounds are quaternary ammonium chloride disinfectants originally designed for sanitation in the dairy, food and restaurant industries. They are potent antimicrobial agents. These compounds have not been registered for use in the treating of fish diseases, but their value as therapeutic agents has been recognized and registration research is in progress.

Hyamine 3500 and Roccal have been marketed in liquid form in concentrations ranging from 10 to 80 percent active ingredients. Hyamine 1622 has been sold in the form of a 100 percent-strength powder which is irritating to the nose and throat. Applicators have been advised to wear a respirator when preparing solutions of Hyamine 1622 and have found that the powder should be added to warm water rather than water to the powder. Hyamine 1622 was found to go into solution easily when added to water but formed a sticky mass if water was poured on the powder.

A standing bath of 2 ppm of the active ingredients of Hyamine 3500 or Roccal, administered for 1 hour, has been used to effectively treat trout for bacterial gill disease. Following treatment the treated water has been replaced with fresh water to prevent over-treating the fish. Lake trout culturists and many salmon culturists have found that Hyamine 3500 was too toxic for safe use in the water available for their use. These workers have successfully used Hyamine 1622 at 2 ppm for 1 hour. In either case, for best results treatments were conducted each day for 3 to 4 days.

 Diquat - Diquat, an "Ortho" herbicide, has been registered for use as an aquatic herbicide in ponds and ditches. It has not been registered as a therapeutic agent for treating fish diseases. Diquat has been sold as a liquid containing 35.3 percent active ingredients. Fish culturists using Diquat to effectively control filamentous algae have found the chemical also controls bacterial gill disease, especially in earthen ponds. The chemical has been expensive and muddy water or heavy accumulations of fish wastes may reduce its effectiveness. Salmon culturists have found it to have low toxicity to salmon fingerlings.

Diquat has been used to treat salmonids at 2 ppm of active Diquat cation (1:500,000) (8.4 ppm as it comes from the bottle) for 1 hour. Applicators have used rubber gloves and respirators to avoid contact with the chemical. Because Diquat is a potent herbicide, fish culturists should avoid using Diquat where the treated water might later be used for irrigating crops.

 Potassium permanganate (KMnO₄) - Potassium permanganate is a strong oxidizing agent that may be of value in some situations. Fish culturists have used it for years to combat Gyrodactylus and other external parasites and it has been of value in some cases of gill disease. Treatment with 3 ppm of KMnO₄ has successfully alleviated the gill proliferation associated with chronically poor water conditions brought on by low flow rates. Potassium permanganate at 2 to 4 ppm for 1 hour on 3 successive days can provide relief. It is suspected that the pond, rather than the fish, is being treated. This procedure has worked, none-the-less. This technique should be further tested wherever low water flow rates lead to gill proliferation.

 Terramycin (TM-50D) - The TM50D formulation of Terramycin is a water-dispersable form which is less costly than some of the other Terramycin formulations that might be added to the water. TM-50D is a Pfizer compound prepared with a sucrose and kaolin carrier which has been used on a limited scale against tail-rot and columnaris. It is used at the rate of 1 ppm and not flushed out of the rearing unit after any certain amount of time. To obtain a 1 ppm final drug concentration, fish culturists have calculated that a 9.1 ppm concentration of the compound, as purchased, is required. No toxicity problems have been reported.

Salt Baths and Dips - Salt is a chemical that is "generally regarded as safe" by regulatory agencies. Not only is salt one of the oldest and most commonly used hatchery chemicals, it is also one of the most overlooked. Even though "miracle" drugs and harsh chemicals seem to be the answer to many fish health problems, sodium chloride or common salt is one of the most effective.

Fish infected with bacterial gill disease, columnaris, or external parasites often generate excessive mucus to combat these disease organisms. This is a natural protective response and many invading organisms are flushed away when this mucus is shed. At the same time, however, this mucus build-up coats the skin and gills covering the parasites or bacteria, shielding them from therapeutic treatments. Successful control of external disease agents is difficult. Salt baths are often helpful to rid fish of excess mucus, physically remove loose parasites and bacteria, and expose remaining organisms to treatment chemicals.

Salt is most effective when used as a 3 percent solution. A 10 to 30 minute treatment period is usually sufficient. If the fish are weak a lower salt concentration and a short treatment period may be necessary but the treatment may have to be repeated again the next day. Fish should not be fed on the days they are being treated.

Salt baths are easily applied in hatchery troughs and tanks by simply shutting off the inflowing water, lowering the water to a suitable treatment level, and adding enough pounds of salt to bring the water to 3 percent salt by weight. Thoroughly dissolve the salt and watch the fish closely. An initial period of excitement among the fish is not unusual. Some fish may lose their equilibrium in salt baths and still not be adversely affected. Care must be taken, however, not to over-treat fish. At the first sign of significant distress or at the end of the 10 to 30 minute treatment period, flush out the salt solution and return the water level and inflow rate to normal.

Constant Flow Treatment

With the exception of salt, the chemicals used in standing baths can be applied by constant-flow siphon devices. The advantage of this method is that the inflowing water is not turned off but is brought to treatment concentration by adding therapeutants to the water. Dense fish populations can be successfully treated in this fashion with a minimum of stress.

Constant flow treatments are administered by setting a known rate of water inflow into the rearing unit and metering a calculated amount of chemical to that inflow to bring it to the desired treatment concentration, usually for 1 hour. The treated water passes through the rearing unit without necessitating lower water levels or special aeration. This method is especially useful in treating eggs for fungus control and in treating fish in raceways and small earthen ponds. Many adaptations of the constant flow technique can be devised to meet local situations. This method is especially useful in ponds and raceways with inflow rates providing less than one water change per hour. The treatment chemical should only be administered for 1 hour, however, and "dead spots" with inadequate water circulation should be specially treated by hand.

Prolonged Treatment

The treatment of fish in large earthen ponds with little or no inflowing water dictates a different approach. Prolonged treatments utilize low levels of chemicals added to the pond water for indefinite periods of time. A desired treatment concentration is selected, the volume of water is calculated, and the appropriate amount of chemical is evenly distributed over the pond. Formalin and copper sulfate are the chemicals most often used in prolonged treatments.

Formalin, at 25 ppm, has been commonly used to control external protozoan parasites. In some situations several treatments at 3 to 5 day intervals are necessary. This treatment should be used with caution if algae blooms are dense, water temperatures are higher than optimum, or oxygen levels are marginal. Copper sulfate, at 0.5 ppm, has been used to control external parasites and also to combat troublesome algae growth. The successful control of stubborn cases of Ichthyophthirius in large ponds containing warm water fish has been reported following the use of a combination of 25 ppm formalin and 0.1 ppm of malachite green. As with other chemicals used in prolonged treatments, the therapeutant was added to the pond water and allowed to dissipate naturally.

When fish populations are dense or phytoplankton blooms are heavy care must be taken not to upset the situation by heavy formalin treatment. In cases of this sort, formalin at 15 ppm may still be effective if repeated at about weekly intervals. Oxygen levels should be monitored and aeration furnished if the formalin treatments could cause significant phytoplankton dieoffs.

Treatments in Transit

The stress of loading fish into distribution units and transporting them to other rearing facilities, to the holding house, or to planting sites can predispose them to bacterial or parasitic infections. If a few diseased fish are present, the entire load will be exposed. Exposure combined with stress could lead to the outbreak of disease. This is especially true in the summer months. Several types of treatments have been helpful.

 Acriflavin at 4 ppm, in distribution tanks, has been used in the transport of warm water fish to combat *Aeromonad* and *Pseudomonad* bacteria which can cause serious delayed mortalities among stressed fish. The water in the tank has been maintained at 4 ppm acriflavin for the duration of the trip.

 Furacin is a nitrofurantoin antibiotic that comes as a "water mix" and has been widely used in the sorting and transfer of bait minnows to inhibit bacterial organisms. It has been used at 5 to 10 ppm of active ingredients for up to 24 hours. Repeated use has not been reported to be harmful to fish.

Salt at 0.5 percent in distribution tanks has been found to greatly reduce hauling losses due to stress and handling injuries in both warm water and salmonid fish culture. The value of salt in the transportation of fish should not be overlooked and this simple, cheap chemical should be evaluated by fish haulers everywhere.

Egg Disinfection

The disinfection of fish eggs began in trout culture because of concern about the spread of furunculosis. There is little evidence that furunculosis can be spread with eggs, however. On the other hand, egg transmission has been demonstrated for IPN and IHN virus diseases and for bacterial kidney disease. Thorough egg disinfection can do much to halt the spread of IHN virus with eggs but disinfection cannot halt the egg transmission of IPN or BKD. Nevertheless, fish culturists routinely disinfect trout and salmon eggs after water-hardening and when they are packed for shipping or unpacked after arrival.

 Betadine, is a trade-name for a complexed iodine compound used in hospitals and elsewhere as a surface disinfectant. It has been sold in the form of a "Prep" or as a "Scrub". The "Scrub" formulation contains a detergent that could increase the toxicity of the compound. The "Prep" formulation has been preferred for salmonid egg disinfection and has been reported to be active against bacteria, viruses, fungi and yeasts. However, if an organism lies within the egg, disinfection cannot affect it without killing the developing fish embryo.

Betadine solution, as it comes from the bottle, contains 1 percent titrable iodine. Other brands of complexed iodine compounds may vary in strength. A treatment concentration of 100 ppm of active iodine has been found to be effective when used in 10 minute treatments. One percent Betadine has been diluted 1:100 to get the desired concentration (1.28 ounces or 38 ml per gallon of water).

A 1:100 dilution of Betadine will acidify soft, poorly buffered water. Sodium bicarbonate (from the kitchen), at the rate of 0.5 grams of baking soda per gallon of disinfectant, has raised the pH to safe levels. Fresh disinfectant solutions were characteristically brown to amber. About 25 ounces of eggs were disinfected per gallon of disinfectant and additional batches of eggs were treated until the solution began to fade to yellow when it lost its strength. Care should be taken in discarding iodine compounds.

The disinfection of either freshly water-hardened eggs or eyed eggs has been successfully carried out in large tubs or shallow troughs with no inflowing water. Aeration of the solution has been considered important, especially if large quantities of eggs were being handled. Upon completion of the 10 minute disinfection period, the eggs were promptly removed and thoroughly rinsed in fresh water. This is an important step, one not to be overlooked, if eggs were to be disinfected prior to shipment. Residual disinfectant could be dangerous to embryos.

 Erythromycin phosphate, at 2 ppm for 1 hour, has been used during the water hardening process for salmon eggs in an effort to halt the egg transmission of bacterial kidney disease. Mixed results were obtained with regard to BKD but the fish could have become infected by other routes. This treatment did, however, result in improved rates of eye-up and improved fry survival. It was speculated that the erythromycin treatment removed contaminating bacteria such as those that cause cold water disease which can cause serious losses shortly after hatching.

Considerable work remains to be done on methods to prevent the egg transmission of serious diseases. It takes a long time to find out if any one method has merit. Avoidance remains the best method of preventing the introduction of many fish diseases.

QUICK REFERENCE CONVERSION TABLE

(Read from left to right)

Unit	Gallon	Quart	Pint	Pound	Ounce	Cubic Ft	Milliliter	Liter	Gram
1 gal	1.0	4.0	8.0	8.345	133.52	0.1337	3785.4	3.785	3785.4
1 qt	0.25	1.0	2.0	2.086	33.38	0.0334	946.36	0.95	946.36
1 pt	0.125	0.5	1.0	1.043	16.69	0.0167	473.18	0.47	473.18
1 lb	0.12	0.48	0.96	1.0	16.0	0.016	453.59	0.454	453.59
1 oz	0.0075	0.03	0.06	0.0625	1.0	0.001	28.3	0.03	28.3
1 cu ft	7.481	29.922	59.848	62.428	998.848	1.0	28317.0	28.317	28317.0
1 ml	0.0003	0.001	0.002	0.002	0.035	--	1.0	0.001	1.0
1 liter	0.264	1.057	2.1134	2.205	35.29	0.0353	1000.0	1.0	1000.0
1 gram	--	--	0.002	0.0022	0.0353	--	1.0	0.001	1.0

Miscellaneous Conversion Factors:

1 kilogram (kg) = 1,000 grams x 2.205 lbs
 1 inch = 2.54 cm or 25.4 mm
 1 cubic foot per second (cfs) = 449 gallons per minute (gpm)
 1 cfs = 646,300 gallons per 24 hours
 1 acre = 43,560 square feet
 1 gram = 15.432 grains
 1 fluid oz = 1.043 oz

From: Wood, James W., 1968, Diseases of Pacific Salmon: Their Prevention and Treatment, State of Washington, Department of Fisheries, Hatchery Division, p. I-4.1.

DISSOLVED OXYGEN SATURATION (ppm) DISTILLED WATER
EXPOSED TO ATMOSPHERE (20.9% OXYGEN GAS) AT
PRESSURES AND TEMPERATURES INDICATED.

<u>ALTITUDE IN FEET</u>				<u>ALTITUDE IN FEET</u>			
<u>Temp.</u> <u>°F</u>	<u>Sea</u> <u>Level</u>	<u>1,000</u>	<u>2,000</u>	<u>Temp.</u> <u>°F</u>	<u>Sea</u> <u>Level</u>	<u>1,000</u>	<u>2,000</u>
32	14.6	14.1	13.5	52	11.0	10.6	10.2
33	14.4	13.9	13.3	53	10.9	10.5	10.1
34	14.2	13.7	13.1	54	10.8	10.4	10.0
35	14.0	13.5	12.9	55	10.6	10.3	9.9
36	13.8	13.3	12.7	56	10.5	10.1	9.7
37	13.6	13.1	12.6	57	10.4	10.0	9.6
38	13.4	12.9	12.4	58	10.3	9.9	9.5
39	13.2	12.7	12.2	59	10.2	9.8	9.4
40	13.0	12.5	12.1	60	10.0	9.7	9.3
41	12.8	12.3	11.9	61	9.9	9.6	9.2
42	12.6	12.2	11.7	62	9.8	9.5	9.1
43	12.5	12.0	11.5	63	9.7	9.3	9.0
44	12.3	11.8	11.4	64	9.6	9.2	8.9
45	12.1	11.7	11.2	65	9.5	9.1	8.8
46	11.9	11.5	11.1	66	9.4	9.0	8.7
47	11.8	11.3	10.9	67	9.3	8.9	8.6
48	11.6	11.2	10.8	68	9.2	8.8	8.5
49	11.5	11.1	10.6	69	9.1	8.7	8.4
50	11.3	10.9	10.5	70	9.0	8.6	8.3
51	11.2	10.8	10.4	75	8.6	8.3	7.9

* Saturation in stream water is usually 0.2 - 0.5 ppm lower than those listed in this table due to dissolved minerals, etc.

From: Wood, James W., 1968, Diseases of Pacific Salmon: Their Prevention and Treatment, State of Washington, Department of Fisheries, Hatchery Division, p. I-4.1.

CONSTANTS and EQUIVALENTS

1 pound	= 16 ounces = 15.25 fluid ounces = 453.59 grams = 7000 grains
1 ounce	= 28.35 grams = 437.5 grains
1 kilogram	= 1000 grams = 2.205 pounds = 35.27 ounces = weight of 1 liter of water
1 gram	= 1000 milligrams = 15.43 grains = weight of 1 cubic centimeter of water
1 gallon	= 4 quarts = 128 fluid ounces = 3.785 liters = 0.134 cubic foot = 231 cubic inches = 3785.43 milliliters or cubic centimeters
1 gallon of water weighs:	8.337 pounds 3.782 kilograms
1 cubic foot of water weighs:	62.364 pounds 28.287 grams
1 second foot	= 1 cubic foot per second
1 cubic foot per second (CFS):	= 448.83 gallons per minute = 26,930 gallons per hour = 646,320 gallons per day = 1795.32 quarts per minute = 107,720 quarts per hour = 57,450 ounces per minute = 101,930 liters per hour = 60 cubic feet per minute = 3600 cubic feet per hour
1 centimeter	= 10 millimeters = 0.394 inches

1 quart = 2 pints
 = 32 fluid ounces
 = 0.946 liters
 = 946.33 milliliters
 = 57.75 cubic inches

1 liquid ounce = 29.573 milliliters
 = 1.805 cubic inches

1 liter = 1000 milliliters or cubic centimeters
 = 0.264 gallons
 = 1.057 quarts
 = 33.815 fluid ounces
 = 61.025 cubic inches

1 liter of water weighs:

1 kilogram
 2.205 pounds

1 milliliter weighs 1 gram

1 cubic centimeter of water weighs 1 gram

1 cubic foot = 1,728 cubic inches
 = 28,317 cubic centimeters
 = 28.316 liters
 = 7.481 gallons
 = 29.922 quarts
 = 957.504 fluid ounces

1 acre = 43,560 square feet
 = 208.7 feet x 208.7 feet
 = a circle whose diameter is 235.504 feet

1 acre foot = 1 acre of surface area 1 foot deep
 = 43,560 cubic feet
 = 2.7 million pounds of water
 = 1233.49 cubic meters
 = 325,851 gallons

1 part per million = 0.0283 grams per cubic foot
 = 1226 grams per acre foot
 = 2.7 pounds per acre foot
 = 0.0038 grams per gallon
 = 0.0000623 pounds per cubic foot

1 percent solution = 38 grams per gallon
 = 1.3 ounces per gallon
 = 0.622 pounds per cubic foot of water

$$\text{Pi} = \pi = 3.1416$$

$$\text{Area of a circle} = \pi r^2 = \frac{\pi d^2}{4}$$

$$\text{Area of a triangle} = \frac{\text{base} \times \text{height}}{2}$$

$$\text{General dilution formula: } \frac{\text{Desired Concentration} \times \text{Desired Volume}}{\text{Concentration of Stock Solution}} =$$

Volume of stock solution to be diluted to desired volume.

POUNDS TO BE ADDED TO OBTAIN DESIRED CONCENTRATION

PPM.	Acre Feet												
	1	2	3	4	5	6	7	8	9	10	11	12	13
.3	.81	1.62	2.43	3.24	4.05	4.86	5.67	6.48	7.29	8.10	8.91	9.72	10.53
.5	1.35	2.70	4.05	5.40	6.75	8.10	9.45	10.80	12.15	13.50	14.85	16.20	17.55
1.0	2.70	5.40	8.10	10.80	13.50	16.20	18.90	21.60	24.30	27.00	29.70	32.40	35.10
2.0	5.40	10.80	16.20	21.60	27.00	32.40	37.80	43.20	48.60	54.00	59.40	64.80	70.20
3.0	8.10	16.20	24.30	32.40	40.50	48.60	56.70	64.80	72.90	81.00	89.10	97.20	105.30
4.0	10.80	21.60	32.40	43.20	54.00	64.80	75.60	86.40	97.20	108.00	118.80	129.60	140.40
5.0	13.50	27.00	40.50	54.00	67.50	81.00	94.50	108.00	121.50	135.00	148.50	162.00	175.50
10.0	27.00	54.00	81.00	108.00	135.00	162.00	189.00	216.00	243.00	270.00	297.00	324.00	351.00
15.0	40.50	81.00	121.50	162.00	202.50	243.00	283.50	324.00	364.50	405.00	445.50	486.00	526.50
20.0	54.00	108.00	162.00	216.00	270.00	324.00	378.00	432.00	486.00	540.00	594.00	648.00	702.00
25.0	67.50	135.00	202.50	270.00	337.50	405.00	472.50	540.00	607.50	675.00	742.50	810.00	877.50

GRAMS TO BE ADDED TO OBTAIN DESIRED CONCENTRATIONS IN PPM's

PPM.	Cubic Feet											
	10	50	100	150	200	300	400	500	1000	1500	2000	3000
.5	.14	.7	1.4	2.1	2.8	4.2	5.7	7.1	14.1	21.2	28.3	42.5
1	.28	1.4	2.8	4.2	5.7	8.5	11.3	14.2	28.3	42.5	56.6	85.0
2	.57	2.8	5.7	8.5	11.3	17.0	22.6	28.3	56.6	85.0	113.3	170.0
3	.85	4.2	8.5	12.7	17.0	25.5	34.0	42.5	85.0	127.4	170.0	254.9
4	1.1	5.7	11.3	17.0	22.6	34.0	45.3	56.6	113.3	170.0	226.6	340.0
5	1.4	7.1	14.1	21.2	28.3	42.5	56.6	70.7	141.6	212.3	283.2	424.6
10	2.8	14.1	28.3	42.4	56.6	85.0	113.3	141.6	283.2	424.8	566.4	850.0
15	4.2	21.2	42.5	63.7	85.0	127.4	170.0	212.4	424.8	637.2	850.0	1274.0
20	5.7	28.3	56.6	85.0	113.3	170.0	226.6	283.2	566.4	850.0	1133.0	1700.0
25	7.1	35.4	70.8	106.2	141.6	212.4	283.2	354.0	708.0	1062.0	1416.0	2124.0

Note: 0.0283 grams in 1 cubic foot of water gives 1.0 ppm.

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